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DISORDERS OF THE SALIVARY GLANDS *

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In the general practice of otolaryngology as well as in the general practice of medicine, one is not infrequently confronted with problems related to disorders in the development and function of the salivary glands. These disorders may be secondary to congenital abnormalities, neoplastic changes, inflammatory conditions, secretory disturbances, or diseases adjacent to the salivary glands. (See Table I).

By salivary glands, we mean not only the major, commonly known, paired glands — the parotid, submaxillary, and sublingual—but also, the aberrantly placed salivary tissue that may be found in the palate, buccal mucosa, lips, and pharynx. Anatomically, this tissue is in close proximity to the oral cavity, and its primary physiology is concerned with it.

The main function of the salivary glands is to produce saliva. The character of the saliva secreted by the submaxillary, sublingual, and parotid glands depends on the type of cell in each particular gland. The parotid secretion is wholly serous; it is thin and watery and has a low content of organic matter. The submaxillary secretion is of the mixed type, mucous and serous. In the sublingual gland, the accini are chiefly of the mucous type with a few serous alveoli. These mucous cells secrete a thick viscid material, rich in mucus. This also is true of the small glands scattered over the buccal mucosa. The type of secretion depends upon the nature of the secretory stimulus, either through mediation of nerve action, chemical influence, drugs, psychic effect, or abnormal metabolic products. Under ordinary conditions, the secretion of saliva is produced by stimulation of the afferent

TABLE 1
DISORDERS OF THE SALIVARY GLANDS
CLASSIFICATION

1. Inflammatory Conditions (Sialadenitis).
   - Nonsuppurative
     - Epidemic and Nonepidemic
   - Suppurative
2. Calculus Formation (Sialolithiasis).
   - Duct
   - Gland
3. Neoplasms.
   - Primary
   - Ectopic
   - Metastatic
4. Secretory Disturbances.
   - Sialorrhea (Increased)
   - Xerostomia (Decreased)
5. Miscellaneous Conditions.
   - (1) Hyperplastic
   - (2) Traumatic
   - (3) Cystic
   - (4) Allergic
   - (5) Anomalous
   - (6) Adjacent structures

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nerves, by food (unconditioned reflex), or by stimulation of some organ of special sense other than taste (conditioned reflex). The saliva is slightly acid in reaction, and in man it amounts to from 1000 cc. to 1500 cc. in a twenty-four hour period, containing 99.5 per cent water and 0.5 per cent total solids. Despite the fact that the parotid gland is the largest of the principal salivary glands, it contributes only 26 per cent of the total salivary secretions. The submaxillary glands, though smaller, produce about two and one-half times as much, or 69 per cent, while the sublingual glands contribute 5 per cent.[1]

Saliva is normally so constantly present in the mouth that we usually fail to appreciate it. The functions of saliva are: (1) action of ptyalin, an enzyme normally present, on the starch molecule; (2) preparation of food for swallowing by altering its consistency to an ingestible gelatinous mass; (3) solvent action to chemically stimulate the taste buds; (4) cleansing action on the lips, teeth, tongue, gums, and oral mucosa; (5) moistening and lubricating the tongue, mouth, and lips; (6) excretion of certain drugs and organic substances; and (7) the regulation of water balance (sensation of thirst).

In addition to these well-known benefits of saliva, there is evidence that other functions may be interrelated with the physiology of iodine, thyroid, and sugar metabolism. Another interesting, more recent use of saliva has been as a prenatal sex test, employing the Rapp-Richardson procedure.[5]

INFLAMMATORY DISORDERS (SIALADENITIS)

In accordance with Table 1, we have classified this disorder as suppurative and nonsuppurative. Enlargement of the salivary glands, whether symptomatic or asymptomatic, may be a prominent manifestation of a general systemic disease or a local one.[2]

The well-known example of the nonsuppurative disorder in this group is epidemic sialadenitis. This is a communicable disease, characterized by swelling, pain, and tenderness, associated with fever. It is a manifestation of a systemic disease, usually occurring in epidemics, and its etiologic agent is known to be a filtrable virus. It may occur in all ages, but is commonly found between the ages of five and fifteen. The course of the illness is self-limited frequently, but one must be acutely aware of its complications, i.e., orchitis, pancreatitis, and encephalitis, with or without inflammatory involvement of the eighth nerve, resulting in permanent deafness. Treatment is primarily that of rest and symptomatic therapy, although many therapeutic agents have been tried without favorable results, including the antibiotics and steroids. There appears to be definite contraindication to the use of the latter in any virus disease.

Another example of the nonsuppurative inflammatory conditions is Mikulicz's disease, a rare malady that is predominantly found in the parotid glands. It is usually asymptomatic, although not invariably so. When symptoms are present, they are mild and not very helpful in distinguishing the disease from parotid neoplasms. Its course is benign, and at present it is thought to be most effectively treated by irradiation.

Brief attention should be directed to Sjogren's syndrome, which has long been recognized by the ophthalmologist as keratoconjunctivitis, but is often not recognized by the otolaryngologist. In our field, it is manifested by atrophy of the mucous membranes of the upper respiratory tract with associated xerostomia and swelling of the salivary glands.

Suppurative sialadenitis is of bacterial origin and may be acute or chronic. The parotid gland is most frequently involved unless the infection is initiated by the presence of a calculus, which is much more often found in the submaxillary glands and ducts. Formerly, it was not uncommon to find suppurative parotitis in elderly patients with a chronic debilitating systemic disease or following major surgery. Since the advent of antibiotics, better

hydration, and the practice of better oral hygiene, the incidence is much lower. The infection is thought to be retrograde from oral sepsis, following decreased parotid secretions caused by dehydration. There is usually unilateral involvement with pain and fever, and on examination the swollen gland is hot and exquisitely painful, and one can frequently express pus through the orifice of Stensen's duct. Treatment is basically that of alleviating the underlying systemic abnormalities, instigating antibiotic therapy, dilatation of the duct, followed by massage of the gland, expressing the purulent material to the orifice of Stensen's duct. Roentgen therapy is not thought to be particularly beneficial in such cases.[1] Rarely now does it become necessary to employ incision and drainage.

Chronic inflammatory conditions of the salivary glands are found more commonly in the submaxillary glands than in the parotid. This is thought to be caused by the higher incidence of calculi or strictures resulting from calculi, with impaired evacuation of secretions. Treatment is surgical extirpation of the gland. In the rarer instances involving the parotid glands, the findings may be bilateral but are most frequently unilateral. The symptoms are recurrent acute attacks of the suppurative process, alternating with periods of relative quiescence. The etiology is unknown, but the condition may be due to failure of a complete remission of an acute attack, or it may be caused by a stricture of the major duct or the lesser radicals, the latter condition sometimes being called sialectasis. This diagnosis has been enhanced by the use of secretory sialography.[6] If the patient is seen during a quiescent period, the involved gland may be slightly larger than normal, and one is able to express viscid purulent secretion in flakes, mixed with clear serous saliva. If stricture is present, it usually can be demonstrated by probing the duct with a lacrimal dilator; and on sialography, one can often demonstrate some abnormality of the duct or gland. Treatment is difficult and not always satisfactory. It consists of repeated dilatations of the duct with massage of the gland, accompanied by intensive specific antibiotic therapy. Occasionally surgical removal of the gland may be necessary to effect a cure.

CALCULUS FORMATION (SIALOLITHIASIS)

Calculus formations in the salivary glands are relatively common, and when present, usually produce symptoms of obstruction. They are more often found in the submaxillary gland and ducts because of the greater viscosity and higher mineral content of these secretions, the longer duct, and the location of the gland at a level inferior to the floor of the mouth, and due to the location of the ostium of Wharton's duct. Stones which are in the duct or in the gland can often be palpated. The symptoms produced are essentially those of obstruction, the first attack being associated with eating, and may be manifested by sudden painful swelling of the gland. The symptoms may subside; but with subsequent attacks, become less severe. Between attacks the gland does not completely return to normal size. As stones are usually present for some time prior to becoming symptomatic, one finds an associated stricture formation that may often cause symptoms to continue after the stone is removed.

Stones are palpable and radiopaque, so that x-ray is an invaluable aid in establishing the diagnosis. A smaller percentage are radiolucent, and then the diagnosis is made by history, palpation—both manually and by means of a probe inserted into the duct — and sialography. When secondary infection is present in the gland, one may express purulent material through the duct. This is more particularly true when the calculus is in the substance of the gland than when in the duct. Treatment of calculi varies with the location. When the stone is in the substance of the gland and has produced obstruction and chronic inflammatory changes, the therapy of choice is excision of the gland. In such situations, one should always be sure that the stone has been removed at the time of surgery and

not pushed distally into the duct. Also, the acute inflammation should be controlled with antibiotics preceding surgery. Calculi in the duct may often be removed through the ostium and delivered into the floor of the mouth. At other times, it may be necessary to dilate the duct with lacrimal dilators and then express the calculus. If one is unsuccessful with these procedures, the duct may be incised, using a dilator or probe as a guide, and then the stone removed with forceps. The latter method has the added advantage of enlarging the stricture.

NEOPLASMS

Neoplasms of the salivary glands have been and are most difficult to classify, as attested by the various classifications one encounters in the literature. More recently, the one presented by Foote and Frazell [3] has been most universally used, and is the one with which we are familiar.

The benign mixed cell tumor is the most common neoplasm encountered in the salivary glands. It is found predominantly in the parotid gland, in a ratio of 10:1 to its occurrence in the submaxillary gland; and, characteristically, the mass has been present over several years without change in size. Historically, it causes no pain and there is no associated facial palsy. Needless to say, clinical differentiation of a tumor between benignancy and malignancy is difficult and treacherous. Pain and facial paralysis are more often experienced in malignancies than in benignancies. The mixed cell tumor is often rounded and unattached to the overlying skin. It is not smooth and on gross examination one sees that it is enveloped in a thin capsule. One should not be misled by this observation at the time of surgery, for microscopically it has been found that strands of tumor extrude through this capsule in the manner of "pseudopods" and project into the apparently normal parotid gland tissue. This explains the high rate of recurrence, 35 per cent, following simple enucleation of the tumor mass, and the present day conception is that mixed cell tumors should be excised with wide margins of normal tissue; in effect, a subtotal parotidectomy should be performed. Every effort should be exerted to preserve the facial nerve at the time of surgery, employing the nerve stimulator freely if necessary. We have not seen a benign tumor involve the facial nerve.

Another example of benign salivary gland tumor that one may find in the parotid is the papillary cystadenoma lymphomatosum or Warthin's tumor. Orloff,[4] in his excellent discussion of this tumor, states that to date no acceptable instance of a malignant transformation of a papillary cystadenoma lymphomatosum has been recorded. Again, surgical therapy rather than irradiation is the treatment of choice.

Mixed tumors may rarely be malignant. However, in spite of this, biopsy of the gland prior to surgery is not recommended. Aspiration biopsy has been employed but not found to be entirely satisfactory, for obvious reasons.

The malignant tumors commonly encountered in the salivary glands, and again much more often found in the parotid, are the squamous cell carcinomas, the adenocarcinomas, and the mucoepidermoid carcinomas. If examination of the tumor at the time of operation causes suspicion of malignancy, a biopsy should be obtained and definitive surgery postponed until an exact diagnosis is established. The treatment of choice is complete extirpation of the gland with sacrifice of the facial nerve, in the case of the parotid, and concomitant en bloc radical neck dissection. If there is doubt that the tumor was or cannot be completely removed by surgery, radiation therapy to tolerance is indicated.

Ectopic salivary gland tumors are not rare. The hard palate appears to be the most common location, but they may occur anywhere in the upper respiratory tract. These tumors are classified according to the ones found in the major salivary glands, and are treated in a similar manner.

Malignancies of adjacent structures, such as the cervical nodes, the ear, and

the pterygomaxillary fossa, may involve the salivary gland by direct extension.

SECRETORY DISTURBANCES

Sialorrhea. (See Table 2).

Excessive salivation may be physiological or pathological, real or apparent. Physiological sialorrhea normally occurs in babies at the time of cutting teeth and

TABLE 2
SECRETORY DISTURBANCES

	Increased (Sialorrhea)	Decreased (Xerostomia)
Physiological—		
	Teething	Exercise
	Foods	
	Tobacco	
Drugs—		
	Iodides	Atropine
	Pilocarpine	Belladonna
	Histamine	Narcotics
	Nicotinic acid	Chlorpromazine
	Reserpine	Dramamine
		Probanthine
		Pathilon
		Antihistamines
Reflex—		
	Emotional stress	Emotional stress
Disease—		
	Oral and pharyngeal lesions	Dehydration
		Irradiation therapy
	Muscle paralysis	Diabetes
	Rabies	Shock
	Paralysis agitans	
	Encephalitis lethargica	
Idiopathic—		
		Elderly females

disappears after the child presents the deciduous teeth. It may be apparent when one is unable to swallow the amount of saliva that is secreted normally, i.e., in obstructive or paralytic lesions of the hypopharynx or esophagus. Physiologic sialorrhea is also present following use of tobacco and as a reflex phenomenon upon the sight or smell of gustatory stimulants.

Pathological salivation is usually found in conjunction with local lesions of the mouth and pharynx, i.e., stomatitis, gingivitis, glossitis, dental disease, malignancies in these areas, and Vincent's infection. In malignancies the sialorrhea is both real and apparent due to difficulty in swallowing. It is also present in emotional stress, rabies, paralysis agitans, and encephalitis lethargica.

The treatment is primarily that of management of the underlying disease. Astringent mouth washes and small doses of atropine may be helpful. In cases of esophageal obstruction or neurological lesions, tracheotomy may be necessary to keep the tracheobronchial tree cleansed of secretions that are aspirated.

Xerostomia. (See Table 2).

Xerostomia or decreased salivary flow may be physiologic or pathologic, temporary or permanent. Physiologically, one finds decreased salivation in dehydration, whether following exercise or underlying systemic disease, and it represents the attempt of the body to conserve fluids. On examination, the mucous membranes of the oral cavity are quite dry, furrowed, and sticky, and the secretions from the salivary ducts are viscid and difficult to express. There are patches of thick tenacious sputum on the posterior pharyngeal wall. Mastication is difficult and one forces food with liquids. Consequently, nutrition is inadequate and this further complicates the picture.

Acquired xerostomia, following radiation therapy to the head and neck, is usually temporary and reversible after completion of the treatment. Certain drugs, i.e., atropine, belladonna, narcotics, antihistamines, and motion sickness medications, also decrease salivation. Emotional stress, too, plays a part, as shown in a public speaker who frequently drinks water during the course of a talk.

Idiopathic xerostomia, found in elderly females, is permanent, irreversible, and quite difficult to treat. Symptomatic and supportive therapy are the primary methods of treatment. Of course, here again, acquired xerostomia must be handled by management of the underlying disease.

MISCELLANEOUS CONDITIONS

The parotid, anatomically, is in an unprotected position and, therefore, vulnerable to trauma. In spite of this, permanent injury to this gland, even in facial injuries, is rare. Temporary fistulae may

develop, but they usually heal in time, even if the larger ducts have been severed.

Allergy may play a part in the development of sialectasis when no evidence of acute or chronic inflammation can be demonstrated in the parotid and submaxillary glands. This is explained by some as being due to obstruction of the radicals by plugs of secretion in which a number of eosinophiles have been noted microscopically.

We have observed at times of stress that there is an idiopathic symmetrical enlargement of the salivary glands, particularly the submaxillary, which is temporary and subsides approximately thirty minutes after the stress reaction has recovered. The etiology of this is not completely understood.

According to Lathrop,[1] congenital anomalies of the salivary glands are rare, but any malformation may develop. These include complete absence of the glands, failure of one or more to develop to maturity, obliteration of the ducts, or formation of cysts. Mention has already been made of aberrant salivary gland tissue.

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CARE AND MANAGEMENT OF THE BURNED PATIENT *

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SHREVEPORT

So much has been written in the past fifteen years about the general and local care of the burned patient that it is worthwhile to reconsider some of the basic principles involved. Much time can be saved, much expense avoided, and far better functional and cosmetic results obtained if a reliable routine plan can be established and followed by those who treat burned patients occasionally in the course of general surgical practice. Such a plan can be used also as a point of departure for critical evaluation of new findings and recommendations as they appear in the medical literature.

A severe burn is a special type of injury, and its treatment is based on the same principles as the management of any other severe trauma—to administer the required supportive treatment, to correct or to avoid a state of actual or impending shock, to protect the wound from further injury, to obtain prompt closure and healing, and to restore normal function and general health as quickly as possible. General treatment and local treatment therefore parallel each other and are conducted simultaneously and with equally close attention throughout the course of the illness. A good final result depends just as much on proper attention to the general requirements of the patient as the local management of the burned areas.

SHOCK

The tendency to development of shock is roughly proportionate to the extent of the burn. In any patient with a burn covering 10 per cent or more of the body surface, the possibility of shock is present, with the probability increasing as the extent of burn increases. This is especially true in children, older people, and patients with coincidental disease such as diabetes, or disease of the heart, lungs, or kidneys, and in patients with associated trauma sustained at the same time as the burn. The possibility of such additional injuries or coincidental disease must always be considered in these patients.

It is well known, of course, that burn shock is due chiefly to sudden and progressive decrease in the circulating blood volume, as a result of the movement of protein-containing fluid from the vascular

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system into the burned areas. This loss of fluid from the blood stream continues for from forty-eight to sixty hours, after which time the resorption of fluid from the tissues back into the blood stream begins. The tendency to shock during this time is increased by the marked loss of erythrocytes from the circulating blood, at least partly due to destruction at the site of the burn. Because of hemoconcentration due to fluid loss, the decrease in red cell mass does not become apparent until after the third day, when plasma loss has ceased and fluid is being restored to the blood stream. With restoration of the normal circulating blood volume after this time, the previously hidden anemia becomes apparent.

The burned patient is seen immediately upon admission and treatment is begun preferably in the operating room, under complete aseptic precautions. The room should be warm rather than cool and drafts or air currents should be kept from the patient, to avoid chilling and discomfort. All burns must be considered to be serious and potentially fatal until proven otherwise and the development of shock must be foreseen in all but minor burns.

IMMEDIATE LOCAL AND GENERAL TREATMENT

The clothing is removed or cut away gently; adherent clothing is soaked off with warm normal salt solution. Tetanus antitoxin is given in the usual prophylactic dosage, after preliminary tests for sensitivity, or a booster dose of tetanus toxoid is given if the patient has been immunized previously. The extent of the burn is estimated as accurately as possible and the patient's weight is estimated or obtained. It is convenient, in estimation of extent of burn, to use the rule of 9, proposed by Wallace [1] and advocated by Evans,[2] in which the skin of the entire head represents 9 per cent of the body surface, each upper extremity 9 per cent, the front of the trunk 18 per cent, the back of the trunk 18 per cent, each lower extremity 18 per cent and the neck 1 per cent, making a total of 100 per cent.

Fluid is given in accordance with the extent of the burn. The formula proposed by Evans [2] is effective and generally applicable. During the first twenty-four hours following injury, blood or plasma or both should be given intravenously in an amount equivalent to 1 cc. per kilogram body weight for each per cent of body surface burned. Whole blood is most satisfactory, both physiologically and because infectious hepatitis has a greater incidence of occurrence following the administration of pooled plasma. If enough blood is not available, dextran solution may be used in the ratio of 1½ to 2 parts of dextran to 1 part of whole blood. Dextran may be used also as a substitute for plasma, but the administration of whole blood is necessary in the significantly burned patient in a proportion of no less than 33 per cent of the total colloid-containing fluid to be given. Not over one liter of dextran solution should be administered in each twenty-four hour period.

The same amount of normal salt solution is given intravenously. In addition, a basic amount of 2,000 cc. of dextrose 5 per cent solution in distilled water is administered in all adult patients, to satisfy the basal requirements for body heat regulation and urinary output. Of this total calculated fluid requirement, one-half is given in the first eight hours following injury and the remaining half in the next sixteen hours.

During the second twenty-four hour period, half this amount of blood or plasma and an equal amount of normal salt solution are given, together with the same basic amount of two liters of dextrose 5 per cent solution in distilled water.

This method of estimation of fluid requirement [2] holds true for burns of up to 50 per cent of the body surface. In more extensive burns than this, or in patients over 50 years of age, the values calculated for burns of 50 per cent of the skin surface are employed. Less than the calculated value is given to patients suffering from cardiac, pulmonary, or renal disease, or from associated burns of the respira-

tory tract, in order to reduce the possibility of pulmonary edema.

When the amount of fluid to be given is large, as in a patient with burns of more than 25 per cent of the body surface, a plastic cannula is inserted into a suitable vein and a constant intravenous drip is established.

Proper regulation of fluid intake during the first two days is achieved by hourly observations of urinary tract output and by frequent determinations of the hemoglobin and hematocrit values. A Foley bladder catheter is introduced and the urinary output is maintained at 35 to 50 cc. per hour. If the secretion of urine varies above or below these limits, the proper correctional adjustments are made in the rate of administration of the intravenous fluids. The hemoglobin and hematocrit values are obtained at the time of admission before fluid administration is begun. These determinations are repeated every four to six hours while the patient is acutely ill and at less frequent intervals after the tendency to development of shock has decreased.

There is no substitute for close personal attention—the patient with burns of more than 25 per cent of the skin surface should be observed hourly during the first forty-eight hours of his hospital stay. After the second day, the fluid which has pooled at the sites of the burns is reabsorbed into the vascular system, and the patient's need for fluid decreases accordingly. At this time, most patients are able to take their basal fluid requirements by mouth, and determinations of the hemoglobin and hematocrit values will indicate if further transfusion is required.

Local treatment of the burn and treatment for actual or threatened shock are carried out simultaneously from the time of the patient's admission. At present, the use of pressure dressings is perhaps the most widely employed method of local management. The burned area is sponged gently with sterile normal salt solution under aseptic precautions, blisters and blebs are opened, desquamated skin layers are trimmed away, the burned surface is flushed again with sterile normal salt solution, and dressings are applied. Sponging with soap is often advocated, but it is not necessary and it may cause pain and irritation of the burned surface. No antiseptics of any kind are employed since all bacteria on the wound surface cannot be destroyed, and infection will be likely to develop on the surface tissues devitalized by the antiseptic.

Several types of dressings are in use. The most commonly employed one unfortunately is a layer of coarse gauze heavily saturated with a thick layer of petrolatum or of Furacin ointment. Such material is entirely unacceptable. The heavy coating of grease will prevent evaporation of moisture from the healing second degree burns, will prevent drainage of exudate from sloughing or infected third degree burns, and will cause maceration of both undamaged and delicate regenerating skin. Much better is a single layer of fine mesh gauze (44 mesh or finer), either dry or impregnated with the thinnest possible film of unmedicated petrolatum. Probably best of all is the use of sterile white rayon or nylon cloth, with a weave fine enough to prevent adhesion even to a granulating surface and yet permeable enough to permit the passage of exudations from the injured surface into the overlying dressings. This cloth is best used dry or dampened simply with sterile normal salt solution.

The local application of powdered sulfonamides and antibiotics or of creams and ointments containing high concentrations of such drugs has been discontinued entirely in recent years, since too many reactions to these compounds have occurred, with both general toxic effects from excessive absorption of the drug and local destructive inflammatory responses on the burned surfaces.

After application of fine mesh cloth to the burn, a layer of flat or fluffed sterile gauze is applied as evenly as possible, then a layer of gauze pads to furnish bulk. The dressing is secured in place

gently with a sterile gauze bandage and wrapped firmly but not tightly with an elastic bandage. Pressure is equalized throughout the dressing; no hard wads of gauze and no tight turns of adhesive tape are permissible since these exert constant local pressure on the burned surface and may cause areas of pressure necrosis.

These dressings can be made up and sterilized beforehand, so that they can be unpacked and applied with no loss of time and with perfect efficiency. Such dressings protect the burn from further injury, afford comfort by providing protection from temperature changes and air currents, and permit epithelization under optimum conditions.

Minor burns of the face and neck are not dressed; extensive burns of these areas are dressed in the routine manner. Whenever it is necessary to include the eyes in a pressure bandage, a light layer of an ophthalmic ointment is applied to the eyes and care is taken that no eyelash is turned onto the cornea. Dressings can be removed, except for the deepest single layer of gauze, from burns of the head and neck after three days, and healing allowed to progress without an overlying dressing. When burns of the digits are dressed, fine mesh gauze or rayon and a thin layer of gauze dressing are placed between adjacent fingers or toes to prevent pressure of each digit upon its neighbor. The fingers and hand are dressed in the position of function. Splints may be applied to the extremities when necessary. These are seldom required because the relatively bulky pressure dressing exerts a satisfactory splinting effect.

Penicillin, either alone or in combination with streptomycin, is given parenterally in the usual dosage to those patients who are able to take these drugs. Others are given erythromycin orally as a satisfactory substitute. The routine use of antibiotics is discontinued after the first five days, since it is unlikely that invasive infections caused by organisms sensitive to these drugs will develop after this long a time, and since the organisms remaining on the burn surface after this time are no longer susceptible to the effects of the drugs.

LATER MANAGEMENT

After seven to ten days, the patient is taken to the operating room and, under aseptic precautions including the use of masks and sterile gloves, dressings are removed down to the final layer of fine mesh cloth. The gauze is dry and clean over areas of second degree burn, is mottled slightly and adherent over areas of mixed second and third degree burn, and is wet and discolored over necrotic areas of third degree burn. The fine mesh gauze is removed only from areas to which it is not adherent, and fresh dressings are applied and allowed to remain for from four to seven days longer.

At the time of the next dressing, twelve to fourteen days after injury, the first and second degree burns have healed and the areas of full thickness burn slough usually are well demarcated. If such areas are present, general anesthesia is induced and the necrotic sloughs or eschars are excised with a sharp scalpel, the plane of excision passing through normal tissue just at the margins of the slough, a good line of cleavage usually being present. Little bleeding occurs as a rule, but if blood vessels of significant size are divided, they are suture-ligated with 5-0 silk or cotton. Bleeding usually can be controlled by pressure, sutures rarely being necessary. The raw surfaces are covered with a layer of fine mesh gauze or nylon, either dry or moistened, and a pressure dressing is again applied. No grease or ointment may be applied to the freshly debrided surface and no medication should be used locally at this time since a film would remain and would interfere with the proper growth of the skin graft when it is applied.

SKIN GRAFTS

On the following day, the patient is returned to the operating room, general anesthesia is induced, dressings are removed under aseptic precautions, and the raw areas are covered with thin split skin

grafts. These grafts are not applied at the time the slough is excised because slight oozing of blood or serum might lift the graft from the raw surface and prevent its growth, and because incomplete excision of the necrotic tissue might not be apparent at the time of debridement but would be obvious the next day. It is probably safer to suture the grafts in place with a running stitch of 5-0 silk around the margin, although this may be omitted if the pressure dressing is applied carefully. If the grafts are sutured, they must not be stretched or sutured under any tension at all, or the graft will fail to take. A skin graft that is not large enough to cover a defect is simply not large enough, and can not be stretched or pulled to fit. A layer of dry fine mesh gauze or nylon is laid over the graft and is moistened with sterile normal salt solution. No grease, ointment, or medication of any type is applied either to the gauze dressing or to the grafted surface. The usual pressure bandage is again applied.

Because such surfaces are always infected, the dressings should be changed after forty-eight hours. By this time, the grafted skin will have become adherent to the raw surface sufficiently to permit careful removal of the fine mesh gauze, irrigation and gentle sponging with warm sterile normal salt solution, removal of any sutures that may have been used, and redressing. On the other hand, if grease gauze has been used, the skin graft tends to become macerated and is much more likely to be dislodged when the dressing is removed. If the first dressing is postponed for more than forty-eight hours, some loss of the graft can be expected, particularly around the margins, from pockets of pus and collections of serum which lift the graft away from the raw surface and destroy its viability in scattered areas. Dressings are changed thereafter every twenty-four to forty-eight hours until the graft is solidly united and dry.

Because of the good blood supply to the skin of the face and neck, it is rarely necessary to excise third degree burn sloughs from these areas, spontaneous separation usually occurring in two weeks. It is especially important to resurface raw areas on the face and neck as early as possible to prevent deformity, contracting scars, and impairment of function of such features as the eyelids. In the same manner, third degree burns involving the hands, feet, axillae, and joint regions must be cleaned and grafted with viable skin within two to three weeks from the time of injury.

It is unwise to wait for spontaneous separation of a burn slough or to delay debridement for more than three weeks at the most. Frequently, also, a granulating surface may become relatively clean and then an encouragingly rapid growth of epithelium may extend inward from all around its margins. It is tempting to allow this to continue in the hope that it will cover the area completely, but the growth soon slows and stops, the granulations lose their healthy red color and become pale, spongy, and dirty, and a dense layer of scar tissue forms beneath the surface. It is perhaps tempting then to apply various ointments or medications or to give the patient various drugs by mouth or by hypodermic injection in the hope that further epithelial growth will be stimulated. This never works; it simply gives more time for further scarring and fibrosis beneath the granulating surface, so that when the too long-delayed graft is finally applied, it is less likely to produce a sound and healthy skin covering, the scar is dense and thick, and the function and comfort of the involved area are permanently impaired. Granulating areas larger than 2 cm. in diameter should be grafted promptly; only areas which are smaller than this should be allowed to heal by spontaneous ingrowth of the epithelium from the edges, and then only if the margins are of normal full-thickness original skin. As Dr. J. Barrett Brown [3] has emphasized, the skin is actually an organ, not simply a covering, and the thin epithelium which grows

outward from normal skin edges to cover over granulating areas is not full-thickness skin but is simply a thin layer of scar epithelium. Such epithelium, situated on a base of dense scar tissue, is fragile and sensitive and never gives a satisfactory functional result. This is true also of the epithelium that fills in the spaces between postage stamp grafts or pinch grafts, and detracts so greatly from the functional and cosmetic results of these types of grafts that it renders them thoroughly unsatisfactory in practically all circumstances. Very thin split-thickness grafts are taken from the donor surfaces, and if good care is taken to protect these regenerating areas, the same donor sites may be used two or even three times at intervals of perhaps three to four weeks. Donor sites from which pinch grafts have been taken will never regain a satisfactory cosmetic appearance and cannot be used later as a source of split-thickness grafts.

OLD INFECTED THIRD DEGREE BURNS

The problem of management of the old infected granulating third degree burn often arises, in which the patient is exhausted, debilitated and anemic, the areas of full thickness burn have deteriorated into surfaces of spongy, pale unhealthy granulations, covered with pus from which a multitude of bacteria of all types can be cultured. Usually these patients already have had every type of antibiotic that is available, and the infecting organisms consequently are resistant to every type of antibacterial drug. Often, too, skin grafts have been attempted, with indifferent success; and some of the surfaces are covered with spotty patches of epithelium, largely of the scar type, which is not really good enough to save but is allowed to remain in the hope that it will eventually cover the interspersed areas of infected granulations. Such surfaces are dressed repeatedly for weeks and months with little progress in epithelization but with much deposition of scar tissue beneath the surface, with permanent impairment of function and unsightly scarring.

General and local measures are both of equal importance in the treatment of such patients, who have lost large quantities of protein from the body tissues. The blood volume and red cell mass must be restored to normal by transfusion of blood, full therapeutic doses of vitamins B complex and C are supplied, with at least 1,000 mg. of ascorbic acid daily, and a high protein diet is given. Although restoration of the blood volume and hemoglobin to normal will improve the patient's appetite, supplementary protein feedings usually will have to be added to the diet. One of the best available means is to give from 3 to 5 ounces of skim milk powder divided into several doses daily between meals, the powder being given either shaken up in water or stirred into liquid skim milk. This type of protein feeding is taken very readily by the patient and is of the greatest value in restoring his nutritional state to normal and in enabling him to grow skin when it is grafted upon a granulating surface.

At the same time, the granulating areas are cleaned by means of daily dressings with the use of dry fine mesh gauze or nylon next to the raw surface. Tub soaks to loosen the dressing before changing are not advisable, because new strains of bacteria are carried through the dressing to the burn surface, adding to the problem of surface infection. The dressings can be changed without pain to the patient simply by soaking them freely in sterile normal salt solution during removal, while coarse mesh gauze dressings cannot be removed without tearing the granulation tissue and causing pain and bleeding. Anesthesia is rarely necessary for change of dressings if rayon or nylon cloth is used. The use of antibiotics, either locally or generally, is unnecessary; within the space of five to seven days and simply by means of daily dressings alone, the surface infection is reduced sufficiently to permit definitive treatment. When the granulations appear clean and pink, the patient is given a general anesthetic, and debridement of the involved areas is performed down to a healthy base. At this

time, the irregular skin margin of the raw areas must be trimmed away, a sharp incision being made through normal skin, with sacrifice of the ragged edge, which is always undermined with pockets of pus and devitalized tissue that will cause necrosis and infection of the margins of the skin graft, with resulting delay in healing and with incomplete take. Split-thickness skin grafts are applied twenty-four hours later.

In these cases too, the grafted areas are dressed after forty-eight hours and at intervals of from twenty-four to forty-eight hours subsequently until full healing has occurred. If dressings are made less frequently, pus pockets will form and will cause patchy necrosis of widespread areas of the grafted skin.

Such frequent dressings require a great deal of time and attention, but for every hour that is spent on proper treatment during the first few weeks of the patient's illness, the doctor and the patient are saved many hours of time and trouble in subsequent weeks or months. Although much time-consuming attention is required daily, a clean burn can be healed with a great deal less time and trouble in its early stages than a dirty granulating old burn in a debilitated patient, which will still require the same daily attention, although for a far longer period and with a far less satisfactory result. It is worth repeating also that an unsatisfactory partial spotty take of a split-thickness graft, with a surface mottled with areas of infected granulations and patches of thin scar epithelium, is not worth saving but should be cleaned, excised in all areas not covered by sound skin, and grafted again.

SUMMARY

In summary, the following points may be emphasized:

1. The development of shock must be anticipated in every patient with significant burns and the proper steps taken to prevent it.

2. Whole blood and plasma or dextran, and crystalloid solutions in amounts calculated according to a safe formula are given in the proper amounts during the first forty-eight hours following burn injury.

3. Adequacy of fluid administration during this time is checked by means of hourly urinary output measurement and frequent hemoglobin - hematocrit determinations.

4. Tetanus antitoxin or toxoid is given in all cases as indicated, appropriate antibiotics are given for the first five days after injury, following which time they are of little value, and large doses of vitamins B complex and C are given during the entire course of recovery.

5. Pressure dressings are applied after the burned areas are cleaned. The dressings are changed first after seven to ten days and again after four to seven days, at which time third degree burn sloughs are excised. The resulting raw surfaces are dressed and covered with thin split-skin grafts 24 hours later. Grafts are dressed for the first time 48 hours after application and at intervals of from one to two days thereafter until healed.

6. No coarse mesh gauze or dressing is ever applied directly to a burn, a granulating surface, or a fresh graft. Thick applications of grease and of antibiotic medications locally are not used. Fine mesh gauze, or preferably white rayon or nylon cloth, is preferred for application to the injured surfaces, either dry or with the barest minimum of sterile petrolatum.

7. Every effort is made to begin the resurfacing of full-thickness burns within two to three weeks after injury, with completion in the shortest possible practicable time. This is especially necessary in third degree burns of the face, hands, feet, genitalia, and in areas around joints.

8. Before skin grafting is done, specific attention is paid to the general nutritional status of the patient with particular reference to restoration of normal hemoglobin values and to a high dietary protein intake, as well as to the proper cleansing of the damaged surfaces.

9. Time spent in close attention to the burned patient in the early weeks saves

a great deal of extra time that would otherwise be necessary to spend in the future.

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INDICATIONS FOR EXPLORATORY THORACOTOMY *

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Exploratory thoracotomy is a diagnostic procedure comparable to exploratory laparotomy. It may and usually does become a therapeutic procedure, but it should be considered one of the aids in the diagnosis of intrathoracic lesions. Such a procedure no longer requires justification since with modern anesthesia, blood replacement and an understanding of pulmonary physiology, the mortality and morbidity are surprisingly low. Hood at the Mayo Clinic reports a 1.3 per cent mortality in 156 patients and a very slight morbidity. An acceptable mortality to be considered is one below 3 per cent.

The clinically obscure pulmonary lesion is an indication for exploratory thoracotomy. These may be considered in the following categories: (1) Mediastinal tumors, (2) atelectasis,, (3) diffuse pulmonary disease, (4) circumscribed lesions of the lungs.

FACTORS IN DIFFERENTIATION

Factors which may be of aid in differentiation of a pulmonary lesion are:

History—The presence or absence of symptoms in itself does not differentiate the lesion, but when present, symptoms are presumptive evidence of a more serious type of pathology. Hemoptysis in the presence of a pulmonary lesion very often indicates a bronchogenic carcinoma, especially in males over fifty years of age.

In the evaluation of a patient with an obscure intrathoracic lesion the diagnostic studies should proceed without delay. If a diagnosis cannot be achieved within a reasonable time, then exploratory thoracotomy is to be considered. In a study of 100 consecutive clinically obscure pulmonary lesions, in which exploratory thoracotomy had been performed, Strug and Beuchner [9] found 50 per cent to be malignant.

Roentgenlogic Examination—It is usually by this means that the disease is found, but not identified. The erect PA of the chest will usually indicate the lesion, but special examinations such as lordotic views, over-penetrated views, stereograms and body section roentgenograms may be necessary. We feel that all patients with pulmonary lesions should have x-rays in both oblique and lateral views.

Bronchoscopy—While most patients with shadows in the lungs should have diagnostic bronchoscopy, the procedure is obviously of little value in the peripheral lesion or in the mediastinal tumor. Occasionally one may obtain either a positive cytological diagnosis by bronchial washings, where the sputum was negative or a bacteriological diagnosis.

Cytological Examination — The usefulness of this study depends on the interest of the pathologist in the Papanicolaou type of cytological staining. The study is a valuable one and those which are positive can no longer be construed as obscure. It is estimated that 60 to 70 per cent of bronchogenic carcinomas will exfoliate cells which will be found in either the sputum or the bronchoscopic washings.[5]

Bacteriologic Examination of Sputum—Acid fast studies of the sputum should be done routinely. Immediate smears, concentrates, and cultures should be performed. Also to be included are examinations for fungus diseases.

Scalene Node Biopsy—Since Daniels de-

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scribed this procedure in 1949,[1] it has become a valuable means both for diagnosis and prognosis. The basic concept is that the mediastinal lymph nodes extend directly into the nodes anterior to the scalenus anticus muscle. It is not necessary that the nodes be palpable in considering a scalene biopsy. The scalene fat pad is excised. In this will be found anywhere from two to twelve small lymph nodes. This is valuable in demonstrating metastatic carcinoma, lymphomas and Boeck's sarcoid. Our experience indicates a lower percentage of positive lymph nodes than that reported by Carr, and Shefts. We have had a recent experience with two patients who had circumscribed peripheral lesions of 3 cms. or less in size, both of whom had a positive scalene node biopsy for carcinoma.

Skin Tests—Intradermal skin sensitivity studies are usually of value in the negative sense. The tuberculin skin test is very rarely negative if the pulmonary lesion is tuberculosis. Those for coccidioidomycosis or histoplasmosis are not quite as important in this negative phase since active lesions of these fungous infections occur with negative skin tests.

SPECIFIC CATEGORIES TO BE CONSIDERED

Mediastinal Tumors—It is rare to be able to diagnose the specific type of lesion when one is confronted with a mediastinal tumor until exploratory thoracotomy is performed. If one sees dentigerous elements in the plain or body section roentgenograms, the diagnosis of dermoid cyst is fairly acceptable. While generalities may be made as to the frequency of different types of tumors in each mediastinal compartment, this is not dependable. In a survey of mediastinal tumors, excluding lymphomas, by Peabody, Strug and Rives[7] the locations and percentages noted were as in Table 1.

TABLE 1
MEDIASTINAL TUMORS

Tumors of the anterior mediastinum:	
Teratoid tumors	20.3%
Thymomas	10.5%
Thyroid adenomas	6.9%
Pericardial coelomic cysts	6.2%
Lipomas	2.0%
Lymphangiomas	1.3%
Tumors of the Posterior Mediastinum:	
Neurogenic tumors	30.6%
Enterogenous cysts	1.1%
Tumors without strong anteroposterior locations:	
Bronchogenic cysts	9.9%
Fibromas	3.7%
Nonspecific cysts	3.4%
Hemangiomas	1.7%

Lymphomas, which include lymphosarcomas and Hodgkin's disease, are predominantly anterior mediastinal lesions and may present on either or both sides of the anterior mediastinum. Here scalene node biopsy may make the diagnosis. Whether or not the tumor presents predominantly on one side or on both of the mediastinum, we feel that exploratory thoracotomy is indicated to establish a histological diagnosis. We no longer feel that a trial of x-ray therapy is justified in such lesions, because of the importance of the specific type of lesion in considering therapy.

Atelectasis—The demonstration of atelectasis by x-ray is evidence only of bronchial obstruction. Unfortunately there are cases of atelectasis which closely resemble atypical pneumonia. If the lesion is lobar in nature, bronchoscopy may lead to an early diagnosis when the clinician is alert to the likelihood of bronchogenic carcinoma. Other lesions which are likely to be manifested by atelectasis are adenoma, foreign body, tuberculous bronchitis, nonspecific granuloma, stenosis of a bronchus, nonspecific pneumonitis and extrinsic pressure on the bronchus. Bronchography can demonstrate the block beyond the reach of the bronchoscope, but cannot give an etiological diagnosis. If segmental atelectasis persists, it is important to consider exploratory thoracotomy since the incidence of bronchogenic carcinoma is so high. Grow and his associates[3] found carcinoma to comprise 31 per cent of the cases of bronchial obstruction.

Diffuse Pulmonary Disease—Within recent years inability to establish a positive diagnosis in some of the diffuse pulmo-

nary diseases, even with our many refined diagnostic measures, has become quite apparent to the majority of individuals interested in pulmonary pathology. These constitute a fairly large number, such as Boeck's sarcoidosis, interstitial pulmonary fibrosis, pneumoconiosis, bagassosis, lymphangitic carcinoma, metastatic carcinoma, and pulmonary adenomatosis.

It is obvious from the roentgenologic characteristics that these diseases cannot, in the majority of instances, be differentiated. The type of prognosis that can be given in such cases depends largely on the accuracy of the histological diagnosis.

With the attendant low mortality and morbidity in exploratory thoracotomy, the risk of entering a thorax and obtaining a biopsy of the pulmonary parenchyma or pleura is negligible. In fact, even in poor risk cases this procedure can be performed under local anesthesia without undue risk.

On our services exploratory thoracotomy for diffuse pulmonary disease is being performed with increasing frequency, and up to the present date with no mortality and minimal morbidity.

Circumscribed Pulmonary Lesions (Coin)—These present our most difficult problem as to the differentiation of an x-ray shadow presenting as a spherical nodule. The term coin is erroneous in that it implies a two dimensional lesion. Actually most of these are more or less spherical in shape. They comprise the following lesions: bronchiogenic carcinoma, sarcoma, bronchial adenoma, metastatic tumors, tuberculomas, histoplasmonas, hamartoma, bronchogenic cyst, coccidioidal granuloma, mesothelioma, lipoid granuloma, A-V aneurysm, and hemangioma.

The difficulty in evaluating these lesions is enhanced by the numerous discrepancies in the literature. Some have stated that small nodules may be ignored, that calcification denotes a benign lesion, that the degree of density may be used to differentiate a benign granuloma from a carcinoma. The statistical evidence is overwhelming that such criteria for differentiation are unreliable, and that exploratory thoracotomy is necessary. Davis, Peabody, and Katz [2] studied 215 such nodules and found 29.7 per cent were carcinoma. Actually 36.7 per cent were malignant. Hood et al [5] from the Mayo Clinic studied 156 cases and found 35.3 per cent malignant. Higginson and Hinshaw [4] found it to be 10.3 per cent in a small series. All agree that solitary lesions of the lung should be treated in the same manner as a mass in the breast. A biopsy must be considered in order to rule out carcinoma.

DIAGNOSTIC CONSIDERATIONS

In the diagnostic considerations to differentiate bronchogenic carcinoma from other lesions, the following factors were evaluated.

1. *History*—This is unlikely to be of any help, unless there is a history of a previous carcinoma elsewhere in the body. Even if the patient had a primary malignancy treated prior to the discovery of the lung mass, one cannot rule out a second primary carcinoma. It is also to be realized that the patient with carcinoma is susceptible to multiple carcinoma, and many reports indicate two or more simultaneous malignancies in the same individual. The diagnostic error may be compounded if one follows the rule that all signs may be explained on the basis of one disease.

2. *Skin Tests*—While it is true that the tuberculin test is quite reliable, we can hardly relate a positive skin test to the specific pulmonary nodule. If the skin test is negative, it is even more urgent to consider the lesion malignant.

3. *Bacteriologic Examination of Sputum*—The solitary tuberculomas rarely produce acid-fast bacilli in the sputum and one cannot depend on this study. It has also been shown that false positives do occur. The concomitant finding of tuberculosis and carcinoma occurs with sufficient frequency to confuse the diagnostic picture. One must be constantly alert to this fact in treating patients with known pulmonary tuberculosis.

4. *Cytology*—A negative report is completely unreliable and a positive report

indicates the need for a therapeutic approach. McBurney [6] in a report from the Mayo Clinic on 29 cases of asymptomatic bronchogenic carcinoma found negative results in 20 of 23 cases studied. Subsequent studies have revealed a higher percentage of positive results particularly in those having squamous cell carcinomas.

5. *Bronchoscopy* — Because of the peripheral nature of these lesions, little can be expected from bronchoscopy. Occasionally one will be able to biopsy such a lesion, or positive cytology may be obtained in bronchial washings. Bronchoscopy proved negative in 20 of 22 cases of asymptomatic carcinoma.[6]

6. *Roentgenologic characteristics*—Size. In Hood's [5] report, no bronchogenic carcinoma was found measuring less than 2 cm. in diameter. On the other hand, 60 per cent of the granulomas were 2.5 cms. or less in diameter. Davis and his associates [2] found 3 bronchogenic carcinomas which were 1 cm. or less in diameter when originally detected; 29 were 3 cm. or less and 41 were between 3 to 5 cms. Size is no criteria of a benign lesion.

7. *Umbilication*—Rigler [8] reported notching of the nodule, a new roentgen sign of malignancy in the solitary pulmonary nodule whether on the conventional film or in the planigrams, invariably meant carcinoma. This only serves to emphasize the urgency of exploratory thoracotomy. What is needed, is a reliable sign which would indicate a benign lesion. There is no such sign.

8. *Calcification*—This is a sign which is constantly debated. Hood [5] reports calcification in 32 per cent of the granulomas and 28 per cent of hamartomas and none in carcinoma. In the calcium free group 20 per cent were bronchogenic carcinoma, 8 per cent bronchial adenomas, and 14 per cent metastatic malignancy. However, there are reports indicating that this is not reliable (Davis and Peabody).[2] In a poll of the members of the American Association for Thoracic Surgery by Davis, 114 members of 328 had encountered a malignant solitary pulmonary nodule containing calcium. Almost all thoracic surgeons agree that punctate calcification of a spotty nature does not rule out bronchogenic carcinoma. Concentric laminations of calcium in a pulmonary nodule is the only reliable sign of a benign lesion. This occurs only in tuberculosis or histoplasmosis. Minimal calcification is not an indication for non-surgical therapy.

CONCLUSIONS

Exploratory thoracotomy as a diagnostic procedure is comparable to exploratory laporatomy.

The indications for exploratory thoracotomy are evident in clinically obscure lesions of the lung. These comprise the following categories: mediastinal tumors, atelectasis, diffuse pulmonary disease, and circumscribed lesions of the lung.

The deficiences in history, roentgenological examinations, bronchoscopy, cytological examinations, bacteriologic examinations, and scalene node biopsy are pointed out in attempts at establishing a positive diagnosis, which further emphasizes the value of exploratory thoracotomy.

The difficulties in diagnosing mediastinal tumors are readily apparent, and the value of exploratory thoracotomy both as a diagnostic and therapeutic procedure is demonstrated.

Atelectasis in itself is only an indication of bronchial obstruction and is not a definite disease entity.

The value of positive information in diffuse pulmonary disease is demonstrated by exploratory thoracotomy, particularly in relation to prognosis.

The various problems in differentiation of circumscribed pulmonary lesions can be resolved by exploratory thoracotomy. The incidence of malignancy in this type of lesion is as high as 49 per cent in reported series. Thus it is obvious that exploratory thoracotomy is both of diagnostic and therapeutic value. In carcinoma localized to the lung, five year survival rates have been reported as high as 36 per cent. It is only by exploratory thoracotomy of the clinically obscure lesions of the lung that we can increase the number of curative

resections in bronchogenic carcinoma.

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CARCINOMA OF THE CERVIX AMONG RESIDENTS OF EAST BATON ROUGE PARISH: 1. INFLUENCE OF SELECTED FACTORS ON MORBIDITY * †

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In studying the epidemiological aspects of cancer of the uterine cervix, comparisons must be made between persons having the disease and those not having it in the same population. Data concerning the race, age, marital status, and parity of persons with carcinoma of the cervix indicate nothing regarding pathogenesis of disease until these factors in the affected persons are compared with the distribution of the same factors in the general population of that area.

This report will deal with the influence of race, age, marital status and parity upon the occurrence of carcinoma of the cervix. It will also show the rate of appearance of this disease in a population. An idea of the rate of occurrence is not altogether of academic interest. The knowledge is necessary in estimating the effectiveness of cancer detection measures and is the starting point in weighing the profit in cancer prophylaxis and treatment. A study of the stage of the disease—both early and late—can give information on the pathogenesis of carcinoma. These questions, then, will be considered regarding this illness: Who is affected by this disease? What is its extent in the population? Can we identify some of the factors influencing its development?

METHOD OF STUDY

The methodological procedures in this report are based on an assumption which from every angle of consideration seems warranted: East Baton Rouge Parish is a fairly large, populous area with a self-contained medical establishment. Nearly all instances of the disease occurring in this population are managed in Baton Rouge and in one other medical facility, Charity Hospital in New Orleans. When diagnosis only is considered, the above observation seems even more sound. It is necessary, therefore, only to analyze the population make-up and to tabulate factors in the disease in question in order to make valid observations on this disease.

Population data were prepared by the Department of Sociology (Institute of Population Research) of Louisiana State University from census figures of East Baton Rouge Parish and the State of Louisiana. Comparisons were made using projections of the population make-up as of January 1, 1954, as this study includes disease diagnosed in the years 1951 through 1956.

Data on the disease under study were

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obtained from the Pelvic Tumor Registry at Our Lady of the Lake Hospital. The Registry was organized by the Baton Rouge Obstetrical and Gynecological Society. A committee was named by the society consisting of two of its members and two pathologists, one from each of the two Baton Rouge hospitals, to set policies and supervise operation of the registry. By consent of all the practicing physicians in the area, every instance of tumor of the female genital organs is recorded in the registry. The operation of the registry is made possible by financial support from the Louisiana Division of the American Cancer Society.

In a further effort to find additional instances of cervical cancer, the death certificates of residents of East Baton Rouge Parish were examined for cancer deaths where the disease had not previously been under medical surveillance. These would be cases presumably where the person affected had not consulted a physician for the illness until the terminal stage was reached. A report from the National Cancer Institute [1] indicates this method of search produced a significant part of all the recorded instances of cancer disease in New Orleans in 1947, 12.5 per cent of the cases. In order to guard against duplication in tabulating total cases, only those cases listed as not recorded prior to death were collected. Their names were then checked against those of patients listed in the Baton Rouge Pelvic Tumor Registry and those of patients admitted to Charity Hospital from this parish.

RESULTS OF THE EAST BATON ROUGE STUDY

Race Factor: Many authors have reported [2-7] that carcinoma of the cervix is more frequent among Negroes, whereas white persons develop more cancers of all sites taken together. For the most part these studies take three forms. Cause of death at autopsy is tabulated in hospital patients and each cause is shown as a per cent of the total, in white and Negro patients. the statement can be made from this type of study: " . . . the percentage of all cancer deaths in the white race is significantly greater . . . than that in the Negro race *in this series of autopsies*." [2] (Italics are ours.) This fact may not be extrapolated to the general population. It is true only for *that series* of autopsies. In another type of report, notably those emanating from the Public Health Service,[1] the analysis is made of data reported to that agency by individual physicians and institutions. Two types of errors can and do occur, omissions and mistakes in diagnosis. However, the large numbers of observations tend to minimize errors of collecting and recording. But in Louisiana, at least, much persuasion must still be done if cancer occurrence is to be accurately reported as a vital statistic by physicians. A third type of report, one which may give a false impression of racial incidence, is that in which a series of cases diagnosed and treated at a given hospital is divided into white and nonwhite groups. Most authors [3-5] reporting this type of study simply cite the ratio of white to Negro patients in those series; however, the reader is tempted to draw the unwarranted conclusion that the ratio holds for the occurrence of the disease in the general population.

In a comprehensive study on this particular question, Warren analyzed 183 cases of carcinoma of the cervix and noted, "Although the nonwhite female rates for . . . cervix uteri were greater than the white rates, the difference [was] not statistically significant." [1]

In the material comprising this report there were 101 instances of cervical carcinoma among white persons and 71 instances among Negroes, a total of 172 cases. Table 1 shows the means by which these cases became known. Table 2 shows the actual number of cases by age group and race. The significant finding is disclosed by the calculation of rates of occurrence by race. There is a definitely higher rate of occurrence in the Negro population than in the white. Figure 1 depicts this fact graphically. There were 34.2 cases of disease per 100,000 nonwhite

TABLE 1
SOURCES OF NEWLY DIAGNOSED CASES OF CARCINOMA OF THE CERVIX, EAST BATON ROUGE PARISH, 1951-1956

Year	Total	Private Patients	Charity Hosp. Patients	Death Certificate
1951	23	9	11	3
1952	32	10	15	7
1953	34	13	21	—
1954	24	14	6	4
1955	28	20	8	—
1956	31	22	6*	3*
Total, 1951-1956	172	88	67	17

* Estimates based on data available for first half of year.

TABLE 2
INSTANCES OF CARCINOMA OF THE CERVIX, BY AGE AND RACE, EAST BATON ROUGE PARISH, 1951-1956

Age	Total	White	Nonwhite
0 - 19	2	—	2
20 - 29	6	3	3
30 - 39	32	12	20
40 - 49	41	29	12
50 - 59	35	25	10
60 - 69	30	17	13
70 & over	26	15	11
Total All Ages	172	101	71

females and 25.2 per 100,000 white females. It does not come within the scope of this report to deal with the reasons for this difference, but only to note that it does exist.

Age Factor: Cancer of the cervix is, naturally, a disease of older persons. But one would wish to know further at what age it occurs most frequently, and whether a woman can "live through" the cancer age, emerging in an era where she would be less likely to have the disease. Is it simply that the older she becomes, the more likely she is to develop cancer of the cervix?

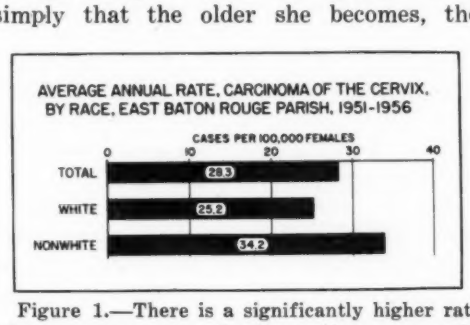

Figure 1.—There is a significantly higher rate of occurrence among the Negro than among the white population.

The experience of others would tend to show that there is a predominance of the disease in the forties and fifties. Robinson [3] in a study not subjected to comparison with population normals states that the peak occurrence is in the fifth decade. Truelson [7] in an exhaustive, highly scientific analysis of age morbidity in 2,918 cases of cancer of the cervix found maximum occurrence in the 45 to 49 year age group. Further, he compared the number of persons having cancer in each age group with the number of females in each age group in the general population, noting that cancer of the cervix occurred 5 times as frequently in the 45 to 49 year group as in the 25 to 29 year group of females; and that it was 3 times as common in the peak age group as in females over 80 years of age.

Age specific morbidity rates presented in Table 3 disclose that in the East Baton Rouge Parish study there was a steady increase of occurrence with advancing age. Although at variance with other studies, there is no doubt about the statistical significance of this observation. Figure 2 effectively portrays this relationship between occurrence and age.

TABLE 3
ANNUAL RATE OF OCCURRENCE OF CARCINOMA OF THE CERVIX (CASES PER 100,000 FEMALE POPULATION), BY AGE AND RACE, EAST BATON ROUGE PARISH, 1951-1956

Age	Total *	White	Nonwhite
0 - 19	0.9	—	2.5
20 - 29	4.7	3.4	7.7
30 - 39	32.5	17.9	63.3
40 - 49	57.6	61.3	50.3
50 - 59	79.9	85.5	68.8
60 - 69	105.0	98.7	114.6
70 & over	147.4	133.7	171.5
Total All Ages	28.3	25.2	34.2

* Based on 172 cases of disease.

Marital Status: Logan [8] reports from a statistical study in England that "marital status alone, apart from child bearing, seems to be a factor in high mortality [from cancer of the cervix]." The study of Wynder [6] revealed that multiple mar-

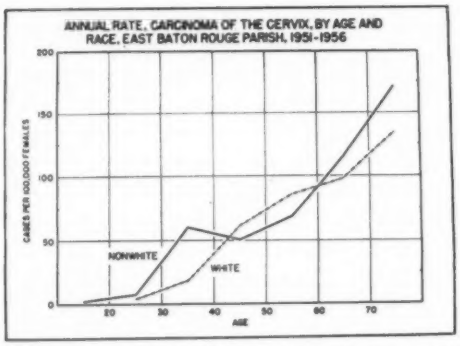

Figure 2.—The incidence of carcinoma of the cervix increases steadily with age.

riages increased the risk to the woman of developing cervical cancer, as did early marriage. But that marriage, coitus, and childbirth are not the only factors involved in genesis of squamous cell carcinoma of the cervix was brought out by Towne,[9] who found 6 instances of the disease in a survey of approximately 13,000 celibate women. Truelson's [7] study showed a somewhat higher morbidity among single women older than 60 years and among separated and divorced women in the respective age groups. It is likely that some factor producing artificial selection accounted for these unexpected and curious findings in Truelson's material.

There are several basic difficulties encountered in evaluating the effect of marital status in the East Baton Rouge study. The prevalence of common law marriages must be considered with accompanying instability of status. There should be due note of the influence of parity and age among married and unmarried persons. And, of course, the factor of pregnancies ending in abortion should be weighed.

The number of instances of carcinoma in the East Baton Rouge Parish study is not large enough to be divided into marital status by age. Hence the disease groups including all ages are compared with population groups of all women 14 years of age and over. This modification makes the "single" category of the control group smaller by eliminating those females who would be expected not to be married because of young age. Census figures are available for this comparison, whereas there are none for marital status in the respective decades of age.

Using the patients' own statement of marital status, Table 4 shows the rate of

TABLE 4
ANNUAL RATE OF OCCURRENCE OF CARCINOMA OF THE CERVIX (CASES PER 100,000 FEMALE POPULATION, 14 YEARS OLD AND OVER), BY MARITAL STATUS AND RACE, EAST BATON ROUGE PARISH, 1951-1956

Marital Status	Total *	White	Nonwhite
Single	4.8	3.7	6.8
Ever Married	38.3	35.2	46.3
Married	39.4	32.1	55.6
Wid. or Div.	32.9	55.2	23.8
All Females	39.1	34.5	48.0

* Based on 141 cases of disease for which marital status is known.

disease per 100,000 females in the categories "single," "married," "widowed or divorced," and "ever married." The principal comparison, between single and ever married, shows an eightfold greater incidence among those married. (Fig. 3) It

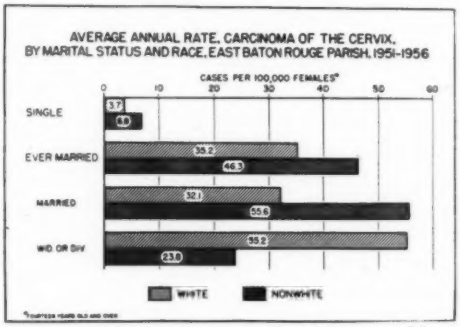

Figure 3.—Married women 14 years old and over develop about eight times as many cervical cancers as single women in the same age group.

is apparent that in the younger age groups there will be more single females and less disease, while in the older groups there will be fewer single females and more disease. However, the magnitude of the difference in rates is sufficient to offset this fact and still remain meaningful.

Parity: Most reports on significance of previous pregnancies in etiology of carcinoma tabulate the number of women having cancer who are parous and the number who are nulliparous. Few authors have attempted to relate these data to the

corresponding breakdown of the total population. Hinselmann [11] calculated a theoretical normal ratio of parous to nulliparous individuals by devious assumptions. He estimated there were 3.2 mothers to each nullipara in the population, and he found the same comparison to be 21 to 1 in patients with cancer of the cervix. Again Truelson, measuring this variable, made careful computations to find the number of nulliparae in a group of healthy females in the same age groups as the patients with cervical carcinoma. He concluded that among 1,000 healthy women of ages corresponding to those of the patients with the disease in question there were 30.6 per cent who had never borne children. Although this normal figure included some women who had had abortions, it is significantly higher than the proportion of nulliparae among his cancer patients, which figured 8.1 per cent. In determining the influence of the number of pregnancies and comparing cancer patients to normal females Truelson noted, "These figures speak against a causal relationship between the number of pregnancies and occurrence of carcinoma of the cervix." [7] And, again: " . . . it is the first pregnancy — and presumably in particular its termination — which is of significance to the occurrence of cervical carcinoma, whereas the number of pregnancies is of no importance." [7]

The Registry material does not contain enough cases of cancer of the cervix at this time to determine disease rates in age groups by number of pregnancies. Also, our basic population data do not provide for a tabulation of number of pregnancies in parous women. The analysis was made simply of parous and nulliparous females with cancer per 100,000 females, 14 years old and over. This information is set forth in Table 5.

According to these rates, proportionately four times as many parous as nulliparous women in East Baton Rouge Parish have carcinoma of the cervix. An interesting observation here is that among nulliparous women ever married there was a higher rate of disease than among single nulliparae. The difference is seen in both white and nonwhite women. (Fig. 4). Since the parity factor does not exert its influence in this comparison, this finding adds greater validity to the conclusion drawn from Table 4. The inference here is that marriage itself has a positive effect upon development of cancer of the cervix.

TABLE 5
ANNUAL RATE OF OCCURRENCE OF CARCINOMA OF THE CERVIX (CASES PER 100,000 FEMALE POPULATION, 14 YEARS OLD AND OVER), BY PARITY AND RACE, EAST BATON ROUGE PARISH, 1951-1956 *

Parity	Total †	White	Nonwhite
Parous	22.4	22.9	21.3
Nulliparous	5.7	6.0	5.1
Ever Married	8.0	8.7	6.9
Single	3.6	3.7	3.4

* Estimates of women ever married with no children are based on 1950 census figures for the State of Louisiana.

† Rates are based on the 72 cases (63 parous and 9 nulliparous) for which parity is known. Two (2) of the parous cases were not identified by race.

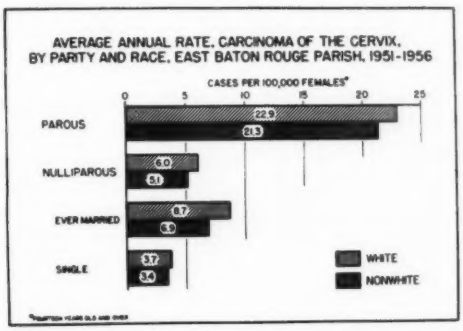

Figure 4.—Rate of occurrence in parous females over 14 years old is four times that in nulliparous females of same age. Among nulliparous married females 14 years old and over, there is twice as much cancer as among single nulliparous women of the same age group.

CONCLUSIONS

1. A study of all known instances of carcinoma of the uterine cervix in East Baton Rouge Parish during the period 1951 through 1956 has been made. There were 172 diagnosed cases.

2. The rate of occurrence is 28.3 cases per 100,000 female population. The rate of occurrence in nonwhites is 34.2. This is significantly higher than the rate of 25.2 in white females.

3. There is a steady increase in rate of occurrence of disease with increasing age —a finding at variance with other research, but found to be significant in this study.

4. Carcinoma of the cervix is proportionately almost eight times as frequent in married women as in single women over 14 years old.

5. Carcinoma of the cervix is approximately four times as prevalent among parous as among nulliparous women.

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SUBACUTE BACTERIAL ENDOCARDITIS: DIAGNOSIS, TREATMENT AND PROGNOSIS *

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LAKE CHARLES

Subacute bacterial endocarditis is a disease which physicians have been generally aware of for only thirty-five years. Marked interest developed concerning this disease following the discovery of antibiotics. Recent reports have added valuable data to the clinical picture and treatment.

A brief review of the literature, with comments on the diagnosis, treatment, and prognosis of bacterial endocarditis will be presented.

CLINICAL FEATURES

I shall discuss this under three headings: (1) general symptoms, (2) signs and symptoms secondary to embolism, and (3) cardiac manifestations.

1. *General Symptoms:*

The onset of subacute bacterial endocarditis is usually insidious but may be acute. The common symptoms listed in their approximate order of frequency are fever, malaise, weakness, lassitude, joint and muscular pains, loss of weight, sweats, chills, and anorexia.

Fever is almost always present. It is irregular and is usually under 103 degrees but in some cases may be high with chills. Patients with bacterial endocarditis may have afebrile periods lasting for days or weeks. This is seen most frequently in patients with bacterial endocarditis of long duration with advanced renal complications. It is also seen in patients who have been treated with only suppressive doses of antibiotics.

In about one-third of the cases the onset of bacterial endocarditis is sudden, marked by fever, chills, and sweats, or by arthritis, or by embolic manifestations such as hemiplegia, Osler's nodes, splenic and renal infarction.

2. *Signs and Symptoms Secondary to Embolism:*

The embolic symptoms are most specific for this disease. Embolic episodes occur at some time in the majority of the cases. It may occur at the onset but usually occurs later. The clinical findings are varied and are dependent on the location of the occluded vessel. The spleen, kidney, and brain are the organs frequently involved, in that order.

The spleen is palpable in about two-thirds of the cases with embolic involvement being most often the cause. The symptoms of splenic infarction may or may not be present.

Renal emboli occur in about 50 per

* Presented at meeting of the Louisiana Heart Association, in Shreveport, La., May 15, 1957.

cent of the cases. Again symptoms may or may not be associated.

With cerebral emboli, neurological manifestations are frequently evident. Various workers state that central nervous system symptoms, secondary to embolic disease, are present in about 15 to 25 per cent of the cases. This aspect of the disease may be so outstanding at the beginning that many of these patients are first admitted to neurosurgical or neuropsychiatric services. The most frequent signs and symptoms associated with cerebral emboli are speech difficulties, vomiting, confusion, tremor, hemiplegia, reflex changes, and cranial nerve palsies.

Pulmonary emboli occur primarily in patients with congenital heart disease who have vegetative lesions on the right side of the heart.[29]

The incidence of retinitis with petechial lesions[30] varies between 3 and 20 per cent. Embolism of the central retinal artery has been estimated to occur in 2.5 per cent.[3] Petechiae involving the skin and mucous membranes occur in the majority of cases.[29]

Osler's nodes and splinter hemorrhages under the nails are frequently seen. There is some debate as to whether or not the latter are of embolic origin.

3. *Cardiac Manifestations:*

The cardiac signs and symptoms are dependent on the severity of the basic heart disease and the degree of involvement of the heart by the endocarditis. A significant murmur is present in 99 per cent of the cases.[3, 12, 30] A mitral systolic murmur is the most common. The changing character of murmurs has been overemphasized.[17] New murmurs may develop. By far the most common is the appearance of an aortic diastolic murmur.

Subacute bacterial endocarditis usually occurs in patients with compensated heart disease.[2] The incidence of congestive heart failure increases with the duration of the disease. The onset of congestive heart failure is usually gradual but may be sudden as a result of rupture of the aortic valve or chorda tendineae.

PHYSICAL FINDINGS

Fever and a heart murmur are almost always present. Splenic enlargement and petechiae occur in 50 to 65 per cent of the cases. Clubbing of the fingers is frequently present. Various workers give an incidence of 35 to 70 per cent[3, 12, 29] Osler's nodes have been noted in 28 to 50 per cent.[3, 12, 30] Neurological findings are present in 15 to 25 per cent. Joints may be hot and swollen in about 10 per cent. Signs of cardiac failure occur in about 10 per cent.

LABORATORY FINDINGS

Mild hypochromic normocytic anemia is usual but it may be marked in the advanced cases. White cell count is normal or slightly elevated. Monocytes may be increased. Phagocytic cells have been noted in the peripheral blood.

Urinary findings: Microscopic hematuria occurs in 50 to 80 per cent of the cases.[3, 12, 30] Albumin, white cells and casts are frequently seen.

Bacteremia: Positive blood cultures occur in 75 to 90 per cent of the cases.[1, 2]

Cerebrospinal fluid abnormalities may be present in cases with cerebral emboli.

Generally Accepted Views on Blood Cultures:

The blood cultures need not be taken at any special time.

A total of five or six blood cultures, collected over a two to three day period is sufficient in the study of a case suspected of having bacterial endocarditis.

Blood should be inoculated on aerobic and anaerobic media. Avoid adding an excessive amount of blood to the culture media since enough natural antibacterial substances may be present, sufficient to interfere with the growth of the bacteria. Venous blood is satisfactory. Cultures should be kept for at least three weeks before they are considered negative.

The physician should have personal contact with the laboratory.

Bone marrow cultures are rarely indicated.

Negative Blood Cultures:

Negative blood cultures occur most fre-

quently in (1) bacterial endocarditis involving the right side of the heart, (2) advanced complicating renal disease, (3) cases who have received antibiotics previously when the disease was not recognized, and (4) in cases in which the enterococcus is the causative agent.[9, 10, 17]

DIAGNOSIS

It is very important that an early diagnosis be made. Subacute bacterial endocarditis must be considered as a serious possibility when a patient with a heart murmur presents with unexplained fever of more than one week.

The classic criteria for the diagnosis of subacute bacterial endocarditis are (1) fever, (2) valvular or congenital cardiac lesion, (3) embolic or vascular lesion, and (4) positive blood culture.

Insistence on the last two criteria accounts for most of the instances of delay in diagnosis and treatment.[12]

The clinical picture of subacute bacterial endocarditis may be so characteristic that the condition can be diagnosed by history and examination alone. The presence of fever, heart murmur, petechiae, splenomegaly, and hematuria allow one to make a diagnosis of subacute bacterial endocarditis immediately.

Sometimes there is a history of tooth extraction or a respiratory infection, two to four weeks prior, to the onset of the symptoms.

One should think of bacterial endocarditis in anyone with congenital or rheumatic heart disease who has unexplained anemia, fever, or heart failure.

While it is easy to describe the signs and symptoms which make up the typical picture of bacterial endocarditis, it is more difficult to state just exactly what are the minimum findings necessary for a diagnosis of this disease.

In some cases, after a complete work-up, only two positive fnidings, an organic heart murmur and unexplained fever of more than one week, are found. Such a patient should be managed as having subacute bacterial endocarditis. Only by accepting these minimal criteria for the diagnosis of subacute bacterial endocarditis can we hope to make an early diagnosis in many of the cases.

It has been stated that the presence of septicemia and murmur alone do not necessarily mean the patient has bacterial endocarditis.[29] However, since it is almost impossible to determine this clinically, one must treat the case as subacute bacterial endocarditis.

One should never rule out the diagnosis of subacute bacterial endocarditis only on the basis of negative blood cultures.

Initial Manifestations which may be confusing:

I shall now outline some of the initial findings in patients with subacute bacterial endocarditis with some comments on how they can lead to confusion with other diagnoses.

Anemia and a cardiac murmur are sometimes the only positive signs noted.[25, 26] The murmur may be erroneously labeled as being hemic in origin.

Confusion with primary neurologic disease may occur, when the patient's illness is manifested primarily with the dramatic suddenness of a cerebral vascular accident. Patients in the younger age group, who present with hemiplegia or subarachnoid hemorrhage and showing a heart murmur and sinus rhythm, should be studied for other evidence of subacute bacterial endocarditis.[18]

Evidence of renal insufficiency may be the most prominent manifestation of this disease.[5, 18, 19, 30] Bacterial endocarditis may be overlooked in the patient who presents with a picture of uremia in which it is thought the renal disease is primary. The debilitated patient with advanced bacterial endocarditis presenting with uremia, anemia, periodic fever, and heart disease, who has been ill for months may be a real diagnostic problem.

Emboli to other sites in the body may draw attention from the primary disease. Thus a renal embolus with chills and fever may be misdiagnosed as acute pyelitis. An arterial embolus in the leg may similarly draw attention away from the primary

disease. A constant awareness of this is the only way one may avoid such mistakes.[30]

Congestive heart failure rarely may be the most prominent manifestation in patients with bacterial endocarditis.

DIFFERENTIAL DIAGNOSIS

Because of the insidious onset of this disease and the multiple organs which may be ultimately involved, many erroneous diagnoses have been made before the true nature of the disease is realized. During the early stages of the disease one may be led to make a diagnosis of virus infection or influenza. Rheumatic fever is frequently considered.

When, after adequate study, the diagnosis of subacute bacterial endocarditis and rheumatic fever still remains in doubt, it is thought best to give the patient a therapeutic trial with penicillin and if he responds to this to continue and treat him as having bacterial endocarditis. If he does not respond then the patient should be given a therapeutic trial with salicylates or steroids.[18]

As a result of the embolic complications, primary cerebral vascular accidents, pyelonephritis, glomerulonephritis, and renal calculus may be considered.[12] MacDonald [20] in a recent report on nonbacterial thrombotic endocarditis noted that this condition, when associated with emboli, may be confused with bacterial endocarditis.

Lupus erythematosus may need to be differentiated.

TREATMENT

It is most important to institute treatment at the earliest possible moment and therefore avoid further damage to the heart and the complications of bacterial endocarditis.

Selection of Drug:

Three factors are very important in choosing a drug as being the ideal therapeutic agent in bacterial endocarditis.[10] (1) It should be bactericidal. (2) It should not be associated with important side effects. (3) It must penetrate clots.

Both penicillin and streptomycin fulfill these requirements. Penicillin is by far the drug of choice in the treatment of subacute bacterial endocarditis. It is generally used in combination with streptomycin. The broad spectrum antibiotics and the sulfonamides are bacteriostatic and do not fulfill the above criteria.[10]

Selection of drug as determined by the offending organism:

A specific etiologic diagnosis is important, not only from the standpoint of making a diagnosis but in administering the proper antibiotic, and in adequate dosage.

Sensitivities of the organisms to antibiotics are generally less important than the past experience with the drugs. Nevertheless, organisms that are recovered from the blood should always have antibiotic sensitivity tests. These tests should measure the bactericidal activity of drugs. The tests for bacterial inhibition (Disk tests) contribute little and may be misleading.

Results of sensitivity tests are usually borne out clinically with penicillin and streptomycin but not so with the broad spectrum antibiotics.[10] The broad spectrum antibiotics frequently only arrest the disease and suppress it only as long as the patient is treated.[10, 22, 27] A combination of penicillin and streptomycin is particularly effective for the *Streptococcus viridans*, and *gamma streptococcus*. There have been reports of apparent cures of subacute bacterial endocarditis with the broad spectrum antibiotics. Nevertheless, it is usually preferable to initiate treatment with penicillin and streptomycin even though the organism is resistant to penicillin. It has repeatedly been shown that penicillin in massive doses has cured patients with organisms which were very resistant to the drug in vitro.

In cases of enterococcus and staphylococcus, which do not respond to massive doses of penicillin and streptomycin, one must then use the drug or drugs which are determined to be best by sensitivity studies.[8] Some of the newer antibiotics such as novobiosin, erythromycin, and oleandomycin have been shown to be of value

in the treatment of resistant staphylococcal infections.

Other drugs, such as bacitracin, neomycin, chloromycetin, and the tetracyclines may be effective.[4, 13, 14] Polymyxin B and neomycin have been used with good results in the treatment of gram negative rods such as pseudomonas, proteus, and *E. coli*, which have become resistant to the usual antibiotics.[8] In the rare case of mycotic endocarditis, amphotericin is the drug of choice.[8]

When to Begin Treatment:

In the clear cut case, enough blood for several cultures is drawn and massive doses of penicillin, along with streptomycin, are immediately started.

In an acutely ill patient, in whom the diagnosis is not too definite, the same method should be followed.

In the mild to moderately ill patient, in whom the diagnosis is not too definite, a period of observation of up to one week is acceptable during which time repeated blood cultures and examinations are performed.

Treatment should not be delayed solely for blood culture reports.

How to Administer Treatment:

When the results of the cultures have not yet been obtained, begin treatment with aqueous penicillin, 6 million units, and streptomycin, 2 grams daily. Various workers start with from 2 million to 12 million units daily.[8, 10, 11, 16] Penicillin is given intramuscularly in divided doses every four hours. Streptomycin is given in one half gram doses every six hours. It is advisable to give half of this as dihydrostreptomycin.

If the patient responds to this treatment then there is no need to change except for reducing the streptomycin to one gram daily after one week. If the patient has not responded, clinically, in two to three days then the penicillin dosage should be increased to 10 million units. If there is no response in another three days then the dose should be increased to 20 million units. The reason for rapidly increasing the dosage is due to the possibility of the offending organism being a staphylococcus or enterococcus, both of which are quite resistant to penicillin and the staphylococcus quickly develops more resistance when inadequate doses of penicillin are administered. If the culture and sensitivity reports are obtained in the meantime then one may alter the medication accordingly.

With patients who respond to the initial doses of penicillin one may switch, after a few days, to the same total dose in the repository form.[18] When penicillin dosage reaches 20 million units daily it is advisable to give this drug continuously intravenously through a polyethylene tube. The penicillin is usually mixed in a total of 1500 ccs. of dextrose in water. Heparin, 25 mgs., is added to each bottle to help avoid the occurrence of thrombophlebitis. Some workers find that, with care, the use of small needles in the veins of the hand or forearm [8] is as good as the use of the polyethylene catheter. Regardless of which method is used it is advisable to change the infusion tubing at two day intervals.

There have been numerous reports concerning the use of renal tubular blocking agents to enhance the blood level of penicillin.[5, 10] The one considered the best at the present time is benemid. Although this drug is effective in achieving this end we have not found any need for it. We think it simpler to increase the dosage of penicillin.

I want to make particular reference to staphylococcal endocarditis. This has become a very difficult therapeutic problem.[10, 28] Not only has there been a marked increase in the incidence of this type of endocarditis, but there has been a marked increase in the percentage of strains of staphylococci which are very resistant to penicillin. In patients in whom the diagnosis of staphylococcal endocarditis is made the initial therapy should consist of even more massive doses of penicillin.[11, 18] The dosage advocated initially has varied from 20 million to 50 million units.[11, 23] Streptomycin is given

as previously outlined.

If there is no response on this regimen in three or four days then the dose of penicillin should be immediately increased. Some workers have given as much as 120 million units of penicillin daily.[23] If the patient is unresponsive to penicillin therapy then the drugs which are best by sensitivity studies are used.[9, 10]

Some reports indicated that two to four of the antibiotics in combination were necessary to effect a cure.[10, 28] Novobiocin, erythromycin, tetracycline or chloramphenicol, either alone or in combination with streptomycin, bacitracin or neomycin, have been used with success.

Accessory therapeutic measures include those which would be indicated in any patient with heart disease and a complicating infection. Absolute bedrest is recommended until the patient is afebrile. Blood transfusions are indicated in the severely anemic.

The steroids and heparin have no place in the therapy of subacute bacterial endocarditis.

Splenectomies have been done in cases of persistent bacteremia.[21] Workers in favor of this procedure suggest that the splenic infarct becomes infected and the antibiotics are unable to penetrate the area.

Duration of Treatment:

In those cases in which the clinical response is immediate and the organism is sensitive to penicillin, it is advisable to continue therapy for three to four weeks after a good clinical response has been obtained. When the organism is not sensitive as in the case of the enterococcus or staphylococcus then give six weeks of additional treatment after clinical response. In any case of relapse do the same.

There are reports [13, 15] on the successful use of short term therapy in patients with penicillin sensitive streptococcus. This consisted of giving penicillin daily for two weeks. The ones who have used this method apparently have had excellent results. This method of treatment has been advised in those cases in whom an early diagnosis is made.

Response to Treatment:

With proper treatment the patient feels better in two to three days. The temperature falls in one to four days and is usually normal in five to seven days. Blood cultures are negative in two to seven days.

Complications of Treatment:

Drug fever: We have seen, not infrequently, a patient who apparently was having a good response to treatment, with the exception of persistent fever. In these cases, stopping the penicillin results in the patient becoming afebrile.

Other sensitive reactions to penicillin are occurring more frequently. In patients who are allergic to penicillin one may try one of the other types of penicillin. If the patient is still sensitive antihistamine preparations or steroids are frequently helpful. The risk of not using penicillin in such cases is greater than the risk of using it.

Thrombophlebitis at the infusion site may give rise to persistent fever.

PROGNOSIS

With the use of antibiotics, a definite change has resulted in the natural history of many infectious diseases.[2] A prime example of this is bacterial endocarditis. This disease, which prior to the use of antibiotics, was almost always fatal, now has a permanent recovery rate of 65 to 70 per cent. It has been shown that such patients have lived up to ten years following treatment.[4, 24] Therefore, the case of healed bacterial endocarditis is a condition that is becoming increasingly important in the natural history of the disease. Today we have the problem of the cured endocarditis patient. A cure of the infectious process is accomplished but sometimes not before it has caused much damage to the affected valve with progressive cardiac deterioration resulting.

The bacteriologic cures are much greater than the survival rate. The patient is cured of the infection but dies from the complications. Christie [6] reports that 35

per cent of successfully treated patients died later. Forty per cent of the cases died from heart failure and 11 per cent from embolic accidents.

Congestive heart failure accounts for the majority of deaths. Renal insufficiency is the second most frequent cause of death.

Factors which affect the prognosis:

Heart failure is a grave sign with the majority of those patients dying during treatment.

Time of treatment: If treated early the prognosis is better. Most workers state that delay in therapy is, at present, the most important cause of treatment failure.[12] Others attach less value to this factor.

Sensitivity of organism to penicillin: Cases in which the causative organism is resistant to penicillin have less chance of recovery.

Negative blood cultures are associated with a higher mortality.[9, 10]

Anemia: Severe anemia is usually a bad sign.

Renal insufficiency. Patients with this complication have a higher mortality.

Cerebral vascular emboli: The prognosis may be altered and is dependent on the degree of damage to the affected area.

Valve involved: The aortic valve, when involved, gives a poorer outlook.[8]

Nutritional state: Those patients with a very poor nutritional state usually have a poorer prognosis.

Age: Because of the more unusual initial manifestations in the aged this disease is recognized later and therefore the outlook is poorer.

Keefer [17] sums up the over-all results of present treatment as follows:

Some patients recover from the infection and remain well and asymptomatic for a period of years. This group makes up 60 to 70 per cent of the cases.

Some recover from the signs of infection and die later of heart failure, uremia, or cerebral embolism. This group accounts for a steady death rate after the first six months, possibly equal to the normal death rate in patients with valvular heart disease.

Patients died during treatment, usually within the first two months, with signs of active infection, heart failure, cerebral embolism or hemorrhage. This group comprises about 10 to 30 per cent of all cases.

Some patients recover from an initial infection only to have reinfection occur at a later date. This group is about 2 per cent.

SUMMARY

A discussion of the diagnosis, treatment and prognosis of subacute bacterial endocarditis has been presented.

Awareness of the typical, as well as the unusual manner of presentation of this disease is essential in order to make an early diagnosis.

Eary diagnosis and adequate treatment are the prime requisites for successful results.

Spectacular advances have been made in the treatment of this disease with the use of antibiotics. However, the mortality rate of 25 to 30 per cent has not been appreciably altered during the past decade.

Therefore, much remains to be accomplished in the management of subacute bacterial endocarditis.

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ASTHMATIC STATES CAUSED BY MUCOVISCIDOSIS *

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NEW ORLEANS

The triad of cough, wheezing and dyspnea signalizes the symptom complex of asthma. Inasmuch as changes that produce asthma are invariably found in the bronchi, it is tautologic to speak of *bronchial* asthma. Nevertheless, asthma is a specific syndrome with characteristic signs, symptoms, and pathologic alterations. By contrast, almost all diseases of the lungs as well as a number of nonpulmonary diseases may produce asthmatic syndromes; in most instances, these conditions can be differentiated by ordinary clinical procedures.

The extreme diversity of conditions that can eventuate in asthma stems from the fact that all involve a bronchial response. In common with other organs there is a limited number of signs and symptoms that can arise from disease or derangement of function of the bronchial tree.

Among the nonallergic conditions that produce asthmatic states is fibrocystic disease of the pancreas (mucoviscidosis). The epoch making work of Dorothy Andersen [3] has delimited this as a specific entity from the broad group of celiac diseases. It is now apparent that meconium ileus in the newborn, and chronic bronchopneumonia in the older infant, are variants of this disorder. Typically, one sees early in the disease repeated, rather severe bouts of infection of the upper and lower respiratory tracts. Evanescent improvement, only, follows use of adequate

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amounts of appropriate antibiotics. With the passage of time the continuing maturation of the disease leads to increasing frequency of such infections, which may be associated with asthmatic symptoms. Review of the records of our 68 children who suffered from this serious systemic disease showed that some degree of wheezing occurred in 33. Thus, a type of intrinsic *bronchial* asthma, or an asthmatic state, may develop in this disorder. Illustrative of this is the following report of cases.

CASE REPORTS

R. M. S., a boy aged 1 year, was admitted to Ochsner Foundation Hospital, in January 1953, because of a persistent infection of the upper respiratory tract. At the age of two months his local physician made a diagnosis of asthmatic bronchitis, which he treated with antibiotics.

Six weeks prior to admission here, pneumonitis developed for which additional antibiotics were given. Since then, the cough and fever had been recurrent. The baby was hospitalized elsewhere in December 1952; on bronchoscopy the mucus membrane of the entire respiratory tree was found to be moderately infected. In the main stem bronchi there was a dry, tenacious secretion from which *Pseudomonas aeruginosa* was cultured

At the time of admission to Ochsner Foundation Hospital the cough and fever were still present. On examination of the chest bilateral moist, crepitant rales were heard. The abdomen was protuberant and the subcutaneous fatty tissue was atrophied. Prominent peribronchial markings having an irregular appearance, consistent with a diagnosis of fibrocystic disease, were noted on roentgenograms of the chest made elsewhere. The stool contained no trypsin, although there was no history of greasy or foul-smelling stools. Material obtained by duodenal intubation contained no trypsin.

D. M., a boy aged 6½ years, was admitted to Ochsner Foundation Hospital because of recurrent infections of the respiratory tract, foul stools, nasal stuffiness and failure to gain weight. Wheezing had been prominent during infancy. This had been followed by repeated infections of the respiratory tract with coughing. During the summer months the patient had frequently complained of a stuffy nose. The stools had always been frothy and bulky with a peculiar odor.

When the patient was first seen by us, his abdomen was protuberant. Both pulmonary fields were clear on auscultation. In the roentgenogram of the chest the peribronchial markings were accentuated bilaterally. The stool contained no trypsin and the duodenal fluid did not digest protein beyond a dilution of 1:16. Pronounced increases of both the sodium and chloride ions were found in the sweat. A nasal smear contained numerous eosinophiles. Reactions to dust, alternaria, and Johnson grass on skin testing were positive.

N. N., a boy aged 20 months, had experienced diarrhea since the third day of life. The fecal discharge was yellow, foul-smelling and foamy. Wheezing had also been apparent early and the infant was taking antihistamines when discharged from the hospital after birth. At the age of 15 months he was treated for his first infection of the respiratory tract. This subsequently reccurred and was associated with a chronic cough.

When the patient was seen at Ochsner Foundation Hospital, loss of subcutaneous fat was obvious. The chest was filled with dry and moist rales and wheezing was clearly audible without the aid of a stethoscope. The duodenal fluid contained no protein digesting enzymes.

DISCUSSION

The mechanical factors known to cause impairment of pulmonary function in asthma are bronchospastic contraction, edema of the mucous membrane and excessive secretion and retention of mucus. The immediate general effect is the same whether this response is the result of allergic factors (allergic bronchospasm, allergic bronchorrhea) or pulmonary infection as is seen in mucoviscidosis; the symptoms and signs may be so similar as to confuse the most experienced clinician. The relative importance of these mechanical factors is not clear; nevertheless they are all present in fibrocystic disease. In point of fact, in the discussion which followed Andersen's paper of 1938, Kramer said, in reference to two children with wheezing and cough, " . . . . the terminal condition began with *asthmatic bronchitis* . . . . ". A group of such children was studied in Canada by Abbott, McCreary and Pocock;[1] 80 per cent had symptoms and signs referable to the respiratory tract; of this group wheezing was heard in 10 per cent, and when present it was noteworthy. Many other instances of asthmatic clinical states have been recorded as occurring in this disease.

What reasons are there for supposing that bronchospasm exists in these unfor-

tunates? On the one hand it has been the common experience of mature pediatricians that administration of epinephrine in such circumstances has led to prompt, albeit transient, alleviation. In addition, in the bronchograms of one of our patients were seen several areas of narrowing of the sort shown by Di Rienzo [12] to be a concomitant of a multitude of pulmonary diseases. The picture shown in Figure 1 is by no means unique; numerous examples of the same sort of

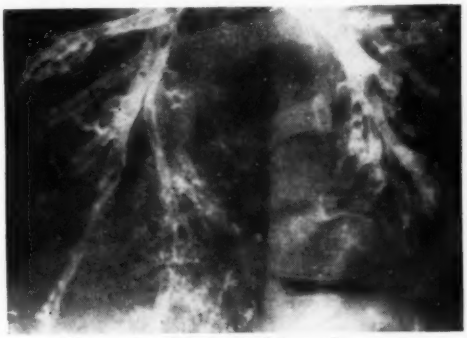

Figure 1. Segmental bronchospasm

segmental bronchospasm can be found in our group of patients with fibrocystic disease of the pancreas.

In Figure 2 we have reproduced evidence of the inflammation and of the

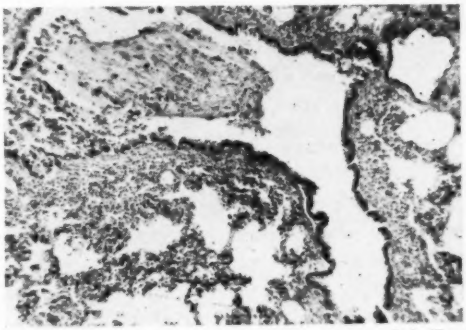

Figure 2. Thick tenacious mucus and inflammation.

thick tenacious mucus so often seen by the pathologist in slides prepared from the lungs of individuals who have died from this disease. Although such mucus has not been studied chemically with the assiduous care applied to investigations of similar material elsewhere in the body, morphologically it does not differ from that seen in other types of cases, notably (according to the leading authority in this field, Andersen) in patients dying during an attack of asthma. Farber,[14] an outstanding pathologist who has studied the disease with notable zeal, commented on the hyperexpansion of the lungs, the result of partial obstruction in the trachea, the bronchi and the bronchioles caused by the thick tenacious mucoid and mucopurulent exudate. He stated, "At times the impression was gained that the patient finally must have experienced suffocation because of the abundance and tenacity of the mucopurulent exudate in the upper respiratory tract."

Attention may be drawn to the bronchoscopic description of Atkins.[5] The expiratory intrusion of the posterior bronchial wall into the lumen produced crescentic cross section to the bronchi not differing, in his opinion, from that seen in asthmatic persons.

SUMMARY AND CONCLUSION

Attention has been focussed on the points of similarity in a significant percentage of patients with fibrocystic disease and in those with bronchial asthma of allergic origin. Because of the malign prognosis of mucoviscidosis, it is the responsibility of all physicians who treat wheezing children to exclude this disorder.

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WATER FLUORIDATION

The artificial fluoridation of water to prevent tooth decay has been proven to be of great value. In our cities generally, technical debate is going on as to whether the fluoride content of public drinking water should be raised as a general health measure. Those participating in the debate are individuals and agencies who advocate this as a public health measure. Opposing fluoridation of water are various interests apparently of the type that in the past have opposed vaccination for smallpox and polio, or chlorination of water as a protection against typhoid fever.

The physician is frequently called upon to express an opinion on this subject which should not continue long as a controversial one.

The research upon this topic has been extensive, has continued longer, and has dealt with greater conclusive statistical evidence than any other public health measure ever presented. The volume of material is such that few could undertake to review it adequately. Dr. Louis I. Dublin, the famed statistician of the Metropolitan Life Insurance Company, has just published a condensation of facts which presents conclusive reasons for fluoridation and answers the specious arguments used by those who oppose it. Dr. Dublin states that ordinarily skepticism is a healthy virtue in public affairs and when a new health measure is first suggested people should have questions about it.

Fluoridation has been introduced and 31,566,000 people have adopted water fluoridation programs. Questions are being raised, and what the thoughtful public needs is facts and not fancies. Facts are presented in the pamphlet in a readable and convincing manner. It is seen that 1506 cities and towns have fluoridation programs now. Chicago, Philadelphia, San Francisco, Pittsburgh, St. Louis, Cleveland, Washington, D.C., Baltimore, St. Paul, Buffalo, Milwaukee, Providence, Miami, and Rochester are among these. Five large cities have rejected fluoridation by referendum and in four others it has been voted down by the city councils. Seventy places, which had inactive water fluoridation programs, have discontinued them.

The facts in support of the use of one part of fluoride per million parts of water are briefly as follows: It is safe and beneficial, and is not productive of any undesirable systemic effects to man. If used from conception through adult life, it reduces tooth decay up to 60 per cent. Its cost is about ten cents per person per year. Fluoride concentrations occurring naturally in water as high as 15 parts per million have not caused bodily harm. The high excess fluorides have caused stained or mottled teeth. No such staining or mottling

occurs when the fluoride is 1.5 parts or less per million of water. Fluoridation is industrially harmless and interferes with no industrial, chemical, or other domestic use of the water.

The objections raised by opponents of fluoridation are effectively answered. It is shown that pure water is a myth. All water contains traces of fluoride. The Supreme Court of Louisiana decided in the case of *Chapman vs. Shreveport* that "addition of fluoride to the water is not medicating it in the generally accepted sense, but was adding to it one of the mineral properties naturally found in water in some sections of the country." Modern city water plants have occasion to add many chemicals to the water furnished. Among these are: activated carbon, fiter alum, ammonia gas or ammonium sulphate, ferrous sulphate, carbon dioxide, chlorine, lime, soda ash, and sodium chloride. Water engineers would have no more difficulty in maintaining the proper amount of fluoride in the water than they have in controlling the amounts of these chemicals already being used.

Alternative methods of using fluoride have been proposed and examined. None has the practical value that fluoridation of the water supply presents. Fluoridation of water has received the greatest support from national scientific bodies of any health proposal ever advanced. These include all the national organizations concerned with health and many who represent civic business and labor interests.

Only about one quarter of the number of Americans who could enjoy better dental health are able to do so through this nutritional measure. Dental health continues to be a matter of great importance. Next to the common cold, tooth decay is the most universal disease suffered by mankind. Dental defects were the single largest cause of rejections among the first two million men examined for military service during World War II. The requirement was that only six upper teeth made contact with six lower teeth. Nearly 10 per cent of the men between the ages of 18 and 35 did not qualify for this reason.

The answer that better dental care should be arranged does not meet the practical needs of the situation. Both dental knowledge and skill are here, but those who need it do not avail themselves of it. Fluoridation will reduce the need up to 60 per cent.

The facts presented in this pamphlet can become a valuable aid to the physician who, of necessity, must have and must express an opinion on the subject, and in matters such as these physicians should continue to take a lead.

Dublin, Louis I.: Water Fluoridation: Facts Not Myths, Public Affairs Pamphlet No. 251, 22 East 38th Street, New York 16, N. Y. Price 25¢.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

CAMERON PARISH MEDICAL REHABILITATION FUND

Everyone is now thoroughly familiar with the death and destruction left behind by Hurricane Audrey which ripped through Cameron and Calcasieu parishes recently, claiming the lives of over 500 people and resulting in countless millions of dollars in property damage.

At the same time, many of you probably are not fully aware of the manner in which our colleagues in the hurricane area acquitted themselves in the period during and after the great disaster.

One veteran disaster worker, Dr. Joseph Hertell of the American Red Cross, commented: "I have witnessed 10 major disasters in the past 5 years. Of all places I have been, the disaster relief operation carried out by the Calcasieu Parish Medical Society was the best planned and executed."

The high praise given by Dr. Hertell is typical of the comments that were heard in the Cameron-

Lake Charles area after the hurricane. All Louisiana doctors should take great pride in the services which our courageous colleagues rendered during this time of great need. Their work was truly in the highest traditions of professional medicine and, in many instances, was far above and beyond the call of duty.

As a tribute to ALL doctors who performed so gallantly during the disaster of Hurricane Audrey, the Louisiana State Medical Society has established a Cameron Parish Medical Rehabilitation Fund which will be used to assist three Society members who were particularly hard hit by the hurricane.

I am referring to Dr. C. W. Clark of Cameron and Drs. G. W. Dix and S. E. Carter of Creole whose physical assets, including their clinics and places of residence, were completely destroyed by the hurricane. In addition, Dr. Clark lost three of his five children.

The newspapers have carried accounts of the heroic contributions which these three doctors made at the height of the hurricane. During and immediately following the storm, they were the only physicians in the areas of worst injury and destruction. It is impossible to fully describe the feats which they performed. They literally worked without sleep for days, and many lives were undoubtedly saved through their efforts.

As far as the Society has been able to determine, the three doctors in question have received no offers of financial assistance to help them rebuild their offices and clinics and to get back into practice. Apparently, this is their most pressing need.

The purpose of the Rehabilitation Fund is to give all members of the Louisiana State Medical Society an opportunity to display their admiration and gratitude to these doctors for their splendid contribution to the cause of medicine.

Contributions of any size will be most welcome.

Because the need is immediate, you are asked to mail checks as quickly as possible (and as large as you can afford) to the following address:

Cameron Parish Medical Rehabilitation Fund
c/o Louisiana State Medical Society
Room 105—1430 Tulane Avenue
New Orleans 12, Louisiana

A.M.A. SPECIAL REPORT OF THE LAW DEPARTMENT MEDICAL PROFESSIONAL LIABILITY

INTRODUCTION

At its meeting in December, 1954, the Board of Trustees requested the Law Department to review previous actions of the American Medical Association with respect to medical professional liability and to plan and initiate any necessary additional studies. This action by the Board was taken in response to a number of resolutions presented to the House of Delegates by state medical societies requesting advice and assistance in this field. After consultation with the staff of the Council on Medical Service and the Committee on Professional Liability of the Committee on Medicolegal Problems, it was determined that further investigation and study was necessary and desirable.

It was recognized at the outset that two approaches to the study were available. We could say as little as possible about the subject for fear of stimulating additional claims or we could plan a program designed to educate the members of the profession concerning accident and claims prevention and alert them to the pitfalls and occupational hazards in the practice of medicine. It was and is our belief that only by facing up to the facts of the past and present concerning medical professional liability can the profession intelligently plan ways and means to cope with this problem in the future.

Since the initiation of its study, the Law Department has submitted three progress reports to the Board of Trustees: one in May, 1955, one in November, 1955, and the most recent in May, 1956. This report is intended to summarize the most significant results of our study up to the present time.

For approximately two years, facts, figures, and opinions have been collected. This material has been reviewed, studied, and analyzed. It is hoped that the results will add to existing knowledge in the field and will provide the basis for workable and effective professional liability claims prevention programs.

SCOPE OF THE STUDY

The following projects have been completed and the results have either been published in THE JOURNAL or will appear within the next few weeks:

(a) State Regulations.—A questionnaire was prepared jointly with the Council on Medical Service and sent to each state insurance commissioner for the purpose of obtaining authoritative information regarding the regulation and control of professional liability insurance rates.

(b) Survey of State Medical Societies.—A questionnaire was sent to all of the state societies and the medical societies of the District of Columbia, Hawaii, and Alaska to obtain the opinion of society officials concerning such subjects as: the average amount of coverage and the availability of professional liability insurance, the most prevalent problems in the field, and the status of claims prevention programs.

(c) State Statutes of Limitation.—A detailed study has been made of the statutes of limitation of each state relating to medical professional liability.

(d) Analysis of Reported Cases.—A review has been made of medical professional liability court cases on which official reports have been published from 1935 through 1955. The analysis of these reported cases indicates the geographical areas in which professional liability cases occur most frequently, the types of medical procedures involved, the circumstances which caused the suits to be filed, and their disposition.

(e) Government Physician.—An analysis has been made of professional liability claims involving physicians in all branches of federal government service.

(f) Survey of National Medical Societies.—A questionnaire inquiring as to available group insurance programs or other similar arrangements was sent to and completed by 13 national medical societies.

(g) Opinion Survey of Physicians.—A questionnaire was sent to approximately 7,500 members of the American Medical Association, representing a random sample of about 5% of the membership. Of these questionnaires 71.2%, or 5,341, were completed and returned. Opinions were requested on various aspects of medical professional liability and inquiry was made as to whether a professional liability claim has ever been brought against them. A second questionnaire requesting detailed information was sent to those physicians who indicated that a professional liability claim or suit has been brought against them.

(h) Special Articles.—The preparation and publication of a series of articles on various aspects of medical professional liability, entitled: The History of Professional Liability Suits in the United States; Expressing Opinions as to Former Treatments; Put It in Writing, Doctor; Medicolegal Hazards of Anesthesia; Hazardous Fields of Medicine in Relation to Professional Liability; Res Ipsa Loquitur—Liability Without Fault; Rule of Respondeat Superior; Professional Liability Insurance; Amount of Coverage; and Professional Liability Claims Prevention.

The above categories of inquiry form the basis for this report. In conducting this study our hypothesis has been that most professional liability claims can be prevented if knowledge of the causes of past claims is put to intelligent use. The information we have obtained, thus far, confirms this belief. Although we have not exhausted all possible sources of information, we have learned a great deal about professional liability and the causes of claims.

THE LAW OF PROFESSIONAL LIABILITY

Although this report is primarily concerned with the legal duty of the physician to avoid injury to his patients, we also of necessity have given some consideration to the physician's ethical, moral, and social responsibilities in the practice of medicine. Generally, the fulfillment of these responsibilities will serve to satisfy the obligations which the law imposes upon the physician.

It is a general rule of law that a physician must possess that degree of medical knowledge and skill possessed by other physicians in his or a similar community engaged in a similar type of practice. He must also use his best judgment and reasonable and ordinary care in applying his knowledge and skill to the treatment of patients. The specialist or the man who holds himself out to the public as a specialist is required to possess and exercise that degree of care and skill commonly possessed by those engaged in the same specialty, in the same or similar community.

THE NATURE OF THE PROBLEM

Patients who have sustained an unsatisfactory result and are aware that they have not received the best possible medical care are potential claimants. Where there is a poor medical result, merely fulfilling legal standards of care is sometimes not enough to prevent a claim. This usually is the case when the patient believes that the physician is not sufficiently sympathetic or if he considers the physician's fees to be excessive.

Professional liability cannot therefore be properly regarded as a legal problem exclusively. It is also a medical problem and one which in our opinion requires the same intensive study that the profession has devoted to the conquering of disease. The legal problems associated with medical professional liability can be dealt with adequately only if medicine will provide the type of emphasis to accident prevention and the utilization of already acquired knowledge as it does to scientific advancement. When effective means are discovered for reducing or minimizing medical professional liability problems it will be physicians who will lead the way by devising techniques that will minimize medical mistakes and patient dissatisfactions.

AVAILABILITY OF PROFESSIONAL LIABILITY INSURANCE AND AMOUNT OF COVERAGE

Without exception, all of the organizational representatives who replied to our medical society questionnaire indicated that medical professional liability insurance was available to the physicians in their states. Furthermore, all of them, except two, stated that it is not difficult to obtain. One indicated that physicians in certain specialties had difficulty, and another said that difficulties had been encountered by physicians who had a previous claim or suit brought against them.

In the survey of individual physicians, 92.3% said that they carried professional liability insurance and 92.6% said that the insurance was not difficult to obtain. Of those answering the questionnaire, 56.4% expressed the opinion that the cost of professional liability insurance is reasonable.

The limits of professional liability coverage appear to vary widely even within a state and within the different types of practice. According to the information supplied by medical society representatives, the average (median) coverage across the country for general practitioners is $25,000 for one claim and $75,000 for all claims during the year; for surgeons and other specialists $100,000 and $300,000. There are at least 45 carriers writing medical professional liability insurance in the United States.

EFFECT OF PROFESSIONAL LIABILITY CLAIMS ON PHYSICIAN'S REPUTATION

A substantial majority of medical society representatives reported that in their opinion professional liability claims have little or no effect on the reputation and on the practice of the physician involved. A few medical society spokesmen explained that in the smaller communities in their area the effects of such claims and suits are more pronounced than in larger communities. Other responses indicated that the effects were greater when newspaper publicity was given to the case. A few responses explained that the effects were more adverse if the physician had previously been the subject of a professional liability claim or suit.

INCIDENCE OF PROFESSIONAL LIABILITY CLAIMS

Many medical society executives and individual physicians have, on numerous occasions in the past, expressed concern over what they describe as an "alarming" increase in the frequency of professional liability claims. It is unfortunate that insurance company records are either unavailable or inaccessible to determine the actual trend. Realizing that the individual physician may not be in a position to supply authoritative information as to whether there is, in fact, a rapid rise in the frequency of claims in his community, in the absence of more accurate data, we nevertheless feel that their opinions deserve consideration. According to our survey of physicians, only 29.7% of the respondents to the question on this point were of the opinion that there has been an increase during the past five years. Thirty-nine and seven-tenths per cent of the respondents felt that the incidence of claims had not increased. The remainder thought that claims had decreased or else they had no opinion.

In California, Louisiana, New York, Rhode Island, Utah, the District of Columbia, and Hawaii, there was a clear-cut expression of opinion that professional liability claims have increased in frequency during the past five years. For example, 59.7% of the California physicians said that in their opinion there has been an increase.

VALIDITY OF CLAIMS

Our study of reported court decisions and the survey of physicians who stated that a claim had been brought against them indicates that approximately 50% of the claims and suits could not be sustained legally. There were, however, a considerable number of instances reported in which a claim was brought against a qualified physician which involved either actual negligence in treatment or substantial basis on which a patient could reasonably believe he suffered from the negligence of a physician. In a few instances it appeared that the claims were either fraudulent or so wholly lacking in foundation as to compel the inference that the patient was acting in bad faith.

Many physicians consider the problems of professional liability as a matter of academic interest. The fact is that professional liability claims are not limited to a small group of "malpractice prone" doctors. Among the physicians who indicated that they had experienced claims, 86.5% incurred only one claim in their entire professional practice. Only 10.5% of the physicians who reported claims had two claims in their entire professional practice; 1.9%, three claims; and 1.1%, four claims.

Our figures indicate that professional liability is the problem of the many, not the few.

In a number of cases which were resolved in favor of the physician because of technical legal grounds, it is possible that the verdict would have been against the defendant had the case been decided on its medical merits. On the other hand, there was a significant number of cases involving the doctrine of "res ipsa loquitur" (the thing speaks for itself) wherein the courts assumed negligence solely because there was no medical explanation for an unsatisfactory result.

PROFESSIONAL LIABILITY CLAIMS REVIEW COMMITTEES

The executives of 31 state medical societies indicated that a claims review program has been established in their state on either a state or county level. The usual procedure followed by these committees is this: When a claim is reported, the physician involved is called in to meet with the committee. The committee attempts to determine whether the claim is legitimate and whether there is evidence of actual professional liability. If the physician has been careless or unethical or has undertaken procedures beyond his competence, he and the insurance carrier are advised to settle the case. If the negligence of the physician is not apparent, every legitimate effort is made to encourage or assist in the defense of the case.

We feel that these committees can render a real service to the public and the profession by indirectly improving the quality of patient care, and in the discouragement of invalid or nuisance claims. Such committees should not attempt to usurp the function of courts in the adjudication of claims nor interfere in the normal relationship between the physician and his insurance carrier.

PROFESSIONAL LIABILITY CLAIMS PREVENTION PROGRAMS

Although only 21 state medical societies reported that they have a claims prevention program, 73.9% of the physicians polled believe that such programs perform a valuable function. Of the physician respondents, 23.7% said that a claims prevention program is now offered by their county medical societies. Of this number, 76.1% rated their county programs as either adequate or excellent.

It appears from these figures, and from the fact that 76.3% of the physicians reported the absence of claims prevention programs in their county medical societies, that there is a nation-wide need and a desire on the part of the medical profession to stimulate the initiation of such programs.

If properly planned and implemented such programs have a twofold objective: the prevention of medical accidents which lead to claims and the prevention of unwarranted claims—in brief, the improvement of medical service.

CLAIMS STATISTICS

Following are some of the significant statistics concerning professional liability claims as shown by our survey of physicians:

(a) 14.1%, or approximately one out of every seven physicians responding to our questionnaire, experienced professional liability claims during his professional medical career.

(b) 53.7% of those who have had claims said that the claims were brought against them since 1950.

(c) 43% said that the alleged act of malpractice occurred since 1950.

(d) 34 years was the approximate median age of patient bringing the claim.

(e) 55% of the claimants were female, but 10 states had more male than female claimants, and 6 states had about the same number of female and male claimants.

(f) 72.5% of the physicians respondents who had claims reported that they had personally performed the treatment or act of alleged malpractice.

(g) 67.2% of the incidents of alleged malpractice occurred in hospitals, 23.9% in the physician's office, 6.3% in the home of patient, and the remaining 2.6% occurred elsewhere in such places as factories, or the place of the accident was not stated by the respondent.

(h) 30.9% of the claims involved surgery, 20.0% medicine, 19.7% orthopedics, 12.5% obstetrics and gynecology, 6.2% neuropsychiatry, 5.6% anesthesiology, and the remaining 1.1% were either too small to tabulate separately or were not stated by the respondent.

(i) The physicians who had 93.2% of the claims reported that they had professional liability insurance at the time of the alleged incident.

(j) 28.9% of the physicians against whom claims were brought are certified by an American specialty board.

(k) 50.4% of the physicians against whom claims were brought stated that they were full-time specialists.

(l) Physicians experiencing claims said that they were in practice, on the average (median), about 13 years before they had a claim.

CONCLUSIONS

After studying the problems of medical professional liability for the past two years, our basic conclusion is that most claims are preventable and not inevitable. We feel that our analysis of professional liability cases and claims and the surveys we have conducted warrant the following specific conclusions:

(a) An element which is present in all professional liability claims is dissatisfaction arising out of the physician-patient relations. Many of the cases which actually involved substandard medical treatment would probably not have matured into claims had it not been for some other cause of friction between the patient and the physician.

(b) Professional liability, although varying in severity in different localities, is a national problem which transcends local boundaries. To be effective, a professional liability claims prevention program requires leadership at the national as well as the state and local levels.

(c) The objective of the medical profession is not the prevention of professional liability claims as such, but the prevention of avoidable errors and omissions that result in injury to the patient and stimulate litigation, and the discouragement of unfounded claims. To implement this objective there is need for (1) an intensive educational program which emphasizes the nonmedical as well as the medical causes for professional liability claims, and (2) the utilization of the self-disciplining resources of the medical profession in the prevention of medical accidents within and outside the hospital.

(d) Regardless of the safety measures that are taken, the ever-increasing complexities of modern medicine create possibilities for human errors and omission even among the most qualified and experienced practitioners.

(e) In the interest of the public as well as the profession, physicians who have demonstrated that they are careless, incompetent or unethical in the treatment of patients should be dealt with effectively through medical society, state licensure and hospital disciplines to prevent the recurrence of patient injury.

(f) An effective educational and accident prevention program should include not only physicians, but physicians' employees and the hospital personnel for whose acts the physician may be responsible.

(g) An effective prevention program should include periodic examinations of equipment to avoid mechanical failures, and the abandonment of obsolete and defective devices.

RECOMMENDATIONS

(a) Considering that more than two out of three of the incidents resulting in professional liability claims occur in hospitals, patient tort liability is now a matter of common interest and mutual concern between the medical profession and hospitals. It is suggested that the Board of Trustees consider the advisability of enter-

ing into discussions with representatives of the American Hospital Association with the objective of formulating and implementing an effective in-hospital safety and accident prevention program.

(b) We recommend that this report be called to the attention of the American Medical Association's representatives on the Joint Commission on the Accreditation of Hospitals for their consideration as to the feasability of encouraging that organization's interest in the subject herein presented.

(c) That state and county medical societies be urged by the Board of Trustees and the House of Delegates to create or, if in existence, implement more effectively, claims prevention programs. To facilitate the efforts of the state societies in this project, the Law Department is forwarding to each state executive secretary all statistics pertaining to his state which have been collected during the course of the current survey.

(d) That state and county medical societies be encouraged to show the film on medical professional liability prevention which will be previewed on June 5, 1957, and to plan informational and educational programs on this subject at state and county meetings.

(e) That the Board of Trustees authorize the printing and distribution of the compilation of medicolegal forms and explanatory text material which has been developed by the Law Department.

(f) That the Law Department be authorized to conduct the second phase of the professional liability survey, consisting of an opinion survey of selected attorneys and the judiciary, an analysis of available information concerning insurance experience and a survey of comparable fields of negligence actions.

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Indepencence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

MAKE ARRANGEMENTS NOW TO ATTEND TRI-STATE MEDICAL SOCIETY MEETING CONFEDERATE MEMORIAL MEDICAL CENTER

Shreveport, Louisiana

September 11-12, 1957

PROGRAM WILL INCLUDE: Both Lectures and Live Clinics

Pediatrics
Dr. Joseph H. Rosenzweig
Hot Springs, Arkansas

Medicine
Dr. Robert Schneider
Oklahoma City, Oklahoma

Orthopedics
Dr. Carrol B. Larson
Iowa City, Iowa

EENT
Dr. M. O. Marchman, Jr.
Dallas, Texas

Obstetrics & Gynecology
Dr. Joseph B. Sheffery
Washington, D. C.

Surgery
Dr. O. H. Beahrs
Rochester, Minnesota

Urology
Dr. Edgar Burns
New Orleans, Louisiana

Pathology
Dr. W. R. Mathews
Shreveport, Louisiana

FELLOWSHIP CERTIFICATES AMERICAN COLLEGE OF CHEST PHYSICIANS

The following members of the Louisiana State Medical Society received their Fellowship certificates in the College at the Convocation on June 1 in New York: Drs. Charles A. Beskin, Baton Rouge; Lawrence Golden, Metairie; Albert L. Hyman, New Orleans; Cheney C. Joseph, Baton Rouge; William Leon, New Orleans, and George A. Skinner, Greenwell Springs.

Dr. Lawrence H. Strug of New Orleans was re-elected Governor of the College for Louisiana.

POSTGRADUATE COURSES ON DISEASE OF THE CHEST

The Council on Postgraduate Medical Education of the American College of Chest Physicians will present the following Postgraduate Courses

on Diseases of the Chest this fall:

12th Annual Postgraduate Course
Hotel Knickerbocker, Chicago, Illinois
October 21-25
10th Annual Postgraduate Course
Park-Sheraton Hotel, New York City
November 11-15
3rd Annual Postgraduate Course
Ambassador Hotel, Los Angeles, California
December 9-13

Tuition for each course is $75. The most recent advances in the diagnosis and treatment of chest diseases — medical and surgical — will be presented.

Further information may be obtained by writing to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

NEW FILM IN A.M.A. SERIES ADVISES DOCTORS HOW TO AVOID PROFESSIONAL LIABILITY HAZARDS

A new film that shows physicians how to avoid the recurrent headache of medical practice today—the professional liability claim—is now available from the American Medical Association film library for county society and other professional bookings.

Titled "The Doctor Defendant", the new film was premiered the week of June 5 in the New York Coliseum before a large audience of physicians attending the 106th annual meeting of the A.M.A. The 34-minute black-and-white sound film is the second in the "Medicine and the Law" film series produced by the Wm. S. Merrell Company, ethical pharmaceutical laboratories of Cincinnati, Ohio, in cooperation with the A.M.A. and the American Bar Association.

The new movie presents in concise and dramatic terms the stories of four doctors who find themselves cast in the disturbing role of "The Doctor Defendant". In reviewing these cases, the film also demonstrates how a county medical society professional liability review committee functions.

Medical societies may book "The Doctor Defendant" from the A.M.A. film library beginning July 1 for showings before their own members.

1957 ANNUAL CONVENTION NATIONAL SOCIETY FOR CRIPPLED CHILDREN AND ADULTS

The 1957 annual convention of the National Society for Crippled Children and Adults—the Easter Seal Society—will be held October 31 to November 2 in Chicago's Palmer House, Dean W. Roberts, M. D., executive director, announced.

Prominent authorities who specialize in the rehabilitation of crippled children and adults as well as lay persons interested in non-scientific aspects of the work will participate in the three-day meeting. Speeches, seminars, workshops, clinics and demonstrations will spotlight the newest techniques and latest information in the care, treatment and training of the crippled.

Delegates expected to attend the meeting will come from Easter Seal societies in the 48 states, District of Columbia, Alaska, Hawaii and Puerto Rico.

Dr. James B. Johnson, prominent Newark, Ohio, orthopedic surgeon, is chairman of the 1957 convention.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

Applications for certification (American Board of Obstetrics and Gynecology), new and reopened, Part I, and requests for re-examination Part II are now being accepted. All candidates are urged to make such application at the earliest possible date. Deadline date for receipt of applications is September 1, 1957. No applications can be accepted after that date.

Candidates for admission to the Examinations are required to submit with their application, an unbound 8½ x 11" typewritten list of all patients admitted to the hospitals where they practice, for the year preceding their application, or the year prior to their request for reopening of their application. This information is to be attested to by the Record Librarian, Superintendent, or Director of the hospitals where the patients are admitted.

Current Bulletins outlining present requirements may be obtained by writing to Robert L. Faulkner, M. D., Secretary, 2105 Adelbert Road, Cleveland 6, Ohio.

UROLOGY AWARD

The American Urological Association offers an annual award of $1000 (first prize of $500, second prize $300 and third prize $200) for essays on the result of some clinical or laboratory research in Urology. Competition shall be limited to urologists who have been graduated not more than ten years, and to hospital internes and residents doing research work in Urology.

The first prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Roosevelt Hotel, New Orleans, Louisiana, April 28-May 1, 1958.

For full particulars write the Executive Secretary, William P. Didusch, 1120 North Charles Street, Baltimore, Maryland. Essays must be in his hands before December 1, 1957.

INTERNATIONAL CONFERENCE OF ULTRASONICS IN MEDICINE

Sponsored by the American Institute of Ultrasonics in Medicine, Statler Hotel, Los Angeles, California, September 6-7, 1957. John H. Aldes, M. D., Secretary, 4833 Fountain Avenue, Los Angeles 29, California.

The meeting will cover the biological and physiological principles, as well as the clinical aspects of ultrasonics in medicine. There will also be a round table conference covering all these phases. Participating in the meeting will be representatives from Europe, South America and Japan.

ACADEMY OF PSYCHOSOMATIC MEDICINE

The program of the fourth annual meeting of The Academy of Psychosocatic Medicine to be held October 17-19, 1957, at the Morrison Hotel in Chicago will be devoted to "Psychosomatic Aspects of Obstetrics, Gynecology, Endocrinology and Diseases of Metabolism". The meeting will be open to all scientific disciplines, as well as psychologists, social workers and nurses. Information may be obtained from Dr. William S. Kroger, Secretary, 104 South Michigan Avenue, Chicago 3, Illinois.

This is the first meeting ever held in this country where every one of these specialties will be presented by a multi-disciplinary approach. The panels and symposiums will include obstetricians, gynecologists, endocrinologists, periodontists, psychiatrists, psychologists, anesthesiologists and other specialists in various fields of medicine. There will also be twenty-four round tables covering all aspects of the above specialties.

The purpose of the Academy is to teach psychosomatic medicine in a manner assimilable to the general practitioner and non-psychiatrically oriented physician.

RADIOLOGICAL SOCIETY ELECTS NEW OFFICERS

In May 1957, the Radiological Society of Louisiana held its annual meeting, and the following new officers were elected:

President, M. Ragan Green, M. D.
Vice-President, J. T. Brierre, M. D.
Secretary-Treasurer, Seymour Ochsner, M. D.

EIGHT HUMAN RABIES DEATHS REPORTED IN 1956

Eight Americans died from rabies in 1956, according to a consultant for the Journal of the American Medical Association.

In addition, one other death was attributed to rabies, but was not confirmed by autopsy, the consultant wrote in the June 29 Journal in response to a query from a Missouri physician.

Four of the confirmed deaths, which were reported to the National Office of Vital Statistics, occurred in Dallas, Texas, in January, June, July and October. The victims were a man aged 63, two boys aged three and 12, and a girl aged eight.

The other deaths occurred in Austin, Texas, in a man 39; Mobile County, Ala., in a woman 26; Lake County, Ind., in a woman 70, and Santa Fe, N. M., in a girl 7.

Rabies, a virus disease, is acquired through the bite of a rabid animal, usually a dog or a cat. Cows, foxes, skunks, bats and other animals also are known to carry the disease. Vaccination of household pets is the best means of stopping the spread of the disease.

According to the National Office of Vital Statistics, the death rates for 1951-55 were: 1951, 18; 1952, 24; 1953, 12; 1954, 13, and 1955, 4.

PICTURES HELP DOCTOR IN EXPLANATION

Visual education has moved into the doctor's office.

Writing in the current (July 13) Journal of the American Medical Association, Dr. John B. Gregg, Sioux Fall, S. D., an otolaryngologist, told how he uses slide films to show patients exactly what is the matter with them.

He has found that interview time can be cut by one third to one half by using pictures. In addition, pictures help the patient to better understand his ailments. In some cases pictures have helped convince patients of the need for operations and have helped allay needless fears, Dr. Gregg said.

He first used pictures torn from medical publications, but they were cumbersome and soon became tattered from use. He next used a hand slide viewer, but this was time consuming if more than one person looked at the slide. Now he projects the pictures on the wall.

In an average office day, the slides are used two or three times, he said.

Educating patients about medical problems is one of the duties of the doctor, but it is frequently neglected because it is so time consuming, he said. The physician-patient relationship breaks down. By using visual education in the office, "greater understanding can be achieved in less time and with less effort," Dr. Gregg concluded.

SABIN SUGGESTS TEST PLAN FOR ORAL POLIO VACCINE

Dr. Albert B. Sabin, Cincinnati, has suggested that counties where the mass use of killed-virus polio vaccine is not economically feasible might be logical place to test his recently developed live-virus vaccine.

In these countries there is no question of waiting to see just how effective the killed-virus (Salk) vaccine will be over a period of years, he said in the current (July 13) Journal of the American Medical Association.

Dr. Sabin, who is with the Children's Hospital Research Foundation, University of Cincinnati College of Medicine, read the article earlier this week at the fourth International Poliomyelitis Conference in Geneva, Switzerland.

His vaccine, a liquid which is taken by mouth, is made with live polio viruses which have been attenuated, or greatly reduced in potency. After the vaccine is swallowed, the viruses multiply in the alimentary tract and cause the body to develop antibodies against the disease.

Recently completed tests on 110 humans showed that a single feeding of attenuated virus produced resistance to reinfection of the alimentary tract comparable to that found in naturally immune persons, while no such resistance was found in people immunized by killed-virus vaccine, Dr. Sabin said.

One of the problems of a live-virus vaccine is the extent to which the viruses excreted by persons after ingestion may be dangerous to other human beings. In fact, this "represents the greatest obstacle to the immediate widespread use of the best attenuated vaccine," Dr. Sabin said.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

The president of the Woman's Auxiliary to the Orleans Parish Medical Society, Mrs. Eugene H. Countiss, called a meeting of the Board members on the morning of May 28th, 1957, at her home on Robert Street for the purpose of expounding and familiarizing each member with the duties and obligations of her chairmanship.

It was an enthusiastic group discussion of many projects and objectives of interest to the Auxiliary.

At the conclusion of the meeting it was evident that a Board of well informed chairmen were eager to assume their responsibilities to the auxiliary and take part in a year of progress, proficiency and pleasure.

A delightful coffee party followed the meeting.

The Woman's Auxiliary will recess during the summer months with the first meeting of the fall season on October 9th at the Orleans Club.

Mrs. Branch J. Aymond,
Publicity Chairman.

BOOK REVIEWS

Anesthesia in Ophthalmology; by Walter S. Atkinson, M. D., Springfield, Illinois, Charles C Thomas, 1955. Pp. 86, Price $3.25. (A monograph in American Lectures in Ophthalmology)

This is probably the most comprehensive, yet concise, discussion of the principles and practices involved in the use of anesthesia in the field of ophthalmology to be found in the English language. For the first time, the physiology, pharmacology and toxicology of local and general anesthetics, and ocular anatomy and neurology are brought together logically and succinctly.

The standard references of Goodman and Gilman, Rovenstine and Adriani, have been combed for basic information. To this has been added the contributions of such pioneers in ocular anesthesia as O'Brien and Kirby. In addition, Dr. Henderson has included some original ideas and procedures, notably a new method of blocking the facial nerves.

About a quarter of the book is devoted to preanesthetic medications and preparation of the patient for surgery. Another quarter is given over to general anesthesia. The rest contains detailed descriptions of topical, infiltration, block, and retrobulbar anesthesia, akinesia, and anesthesia of the lids, lacrimal sac and conjunctiva. Drugs, dosages and equipment, down to length of needles and size of syringes, are conveniently listed. Controversial subjects like curare and hyaluronidase are treated in separate chapters. The diagrams and pictures are excellent, and the illustrations for the chapter on anesthetic emergencies bring home vividly the signs of procaine reactions and their treatment.

The material on general anesthesia is written from the viewpoint that "the importance of having a competent anesthesiologist (give the anesthetic) cannot be overemphasized". Frequently in everyday practice a nurse anesthetist must be used in his place and consequently any serious untoward reaction or complication must be attended to by the surgeon. It is for this reason that a description of cardiac arrest and its treatment might well have been included.

Editorially, the publisher would do well to proofread again pages three and ten for word and sentence structure. However, these are minor printing errors and do not detract from the importance of Dr. Atkinson's fine work. This

classic monograph deserves to be read and studied by every resident and practicing ophthalmologist.

JOSEPH P. RUMAGE, M. D.

Causal Factors in Cancer of the Lung; by Carl V. Weller, M. D., Springfield, Illinois, Charles C Thomas, 1956, Pp. 113, $3.00.

In this brief and well organized monograph based upon the Beaumont Lecture for 1955, Dr. Weller, an eminent pathologist, has presented the evidence which implicates extrinsic factors in the production of bronchogenic carcinoma. Before considering external causation, he points out that no hereditary patterns for this neoplasm have been demonstrated; this makes the existence of important extrinsic factors even more likely and improves the outlook for ultimate prevention of this common and deadly disease. Specific external factors are introduced by expert use of the historical method. Carcinoma of the lung is considered in relation to occupation, atmospheric pollution, and tobacco smoking.

The evidence is impartially collected and the author's opinions are expressed at the close of each section. He concludes that there is a definite association between the processing of chromates and asbestos and lung cancer. Mining at Schneeberg seems causally related to bronchogenic carcinoma but specific factors are not yet established; radioactivity and arsenic, acting alone or together, are the likeliest agents. The role of carcinogens in the atmosphere of modern cities requires further investigation.

Dr. Weller concludes his discussion of tobacco smoking with agreement that the association of heavy cigarette smoking and lung cancer has been established. He hesitates to personally advise the medical profession concerning preventive measures but ends his book with a quotation which counsels avoidance of heavily implicated extrinsic factors because it may be many years before the specific carcinogens necessary for direct proof are discovered.

This volume is recommended to all physicians and students of medicine.

MORTON M. ZISKIND, M. D.

Peripheral Vascular Diseases; by Edgar V. Allen, Nelson W. Barker and Edgar A. Hines, 2nd Edition, Philadelphia, Pa., W. B. Saunders Company, 1955, Pp. 825, Illus. 316. Price $13.00.

This book covers diseases of blood vessels and lymph vessels. It is well written and illustrated and includes diagnostic methods and both medical and surgical therapy as well as some pathophysiological and historical background discussions. It is recommended for the student and practising physician as a ready reference book of diagnostic criteria and techniques and definitive therapy.

CHARLES C. ABBOTT, M. D.

PUBLICATIONS RECEIVED

Alomaeon Publications, N. Y.: Opinions and Critical Judgments on Prof. G. P. Arceri's book, The Circulation of the Blood and Andrea Cesalpino of Arezzo.

Appleton-Century-Crofts, Inc., N. Y.: Practitioners' Conferences, Volume 6, edited by Claude E. Forkner, M. D.

Group for the Advance of Psychiatry, N. Y.: Psychiatric Aspects of School Desegregation, formulated by the committee on social issues.

Grune & Stratton, N. Y.: Gout, by John H. Talbott, M. D.; Essentials of Clinical Proctology, by Manuel G. Spiesman, M. D., and Louis Malow, M. D. (3rd edit.); Practical Refraction, by Bernard C. Gettes, M. D.

Little, Brown & Co., Boston: Martius' Gynecological Operations, With Emphasis on Topographic Anatomy, translated and edited by Milton L. McCall, M. D., and Karl A. Bolten, M. D., foreword by Edward A. Schumann, M. D. (7th edit).

The C. V. Mosby Co., St. Louis: Synopsis of Obstetrics, by Jennings C. Litzenberg, M. D., revised by Charles E. McLennan, M. D. (5th edit.); Atlas of Clinical Endocrinology, by H. Lisser, M. D., and Roberto F. Escamilla, M. D.; Modern Therapy in Neurology, edited by Francis M. Forster, M. D., with foreword by H. Houston Merritt, M. D.

Philosophical Library, N. Y.: William Harvey, His Life and Times: His Discoveries: His Methods, by Louis Chauvois; Epilepsy, Grand Mal, Petit Mal Convulsions, by Letitia Fairfield, M. D.

W. B. Saunders Co., Phila.: Textbook of Pathology, With Clinical Applications, by Stanley L. Robbins, M. D.; Therapeutic Exercise for Body Alignment and Function, by Marian Williams, Ph.D., and Catherine Worthingham, Ph.D.; The Principles and Methods of Physical Diagnosis, by Simon S. Leopold, M. D. (2nd edit.); Gifford's Textbook of Ophthalmology, by Francis Heed Adler, M. D.; (6th edit.); The Treatment of Burns, by Curtis P. Artz, M. D., and Eric Reiss, M. D.; A Textbook of Histology, by Professors Alexander A. Maximow and William Bloom (7th edit.).

Stanford University Press, Stanford, Calif.: Health Yearbook, 1956, compiled by Oliver E. Byrd, M. D.

Charles C Thomas, Publisher, Springfield, Ill.: Magnetic Removal of Foreign Bodies, by Murdock Equen, M. D.; The Diagnosis and Treatment of Endocrine Disorders in Childhood and Adolescence, by Lawson Wilkins, M. D. (2nd edit.); The Early Diagnosis and Treatment of Acoustic Nerve Tumors, by J. Lawrence Pool, M. D., and Arthur A. Pava, M. D.; Blood Transfusion in Clinical Medicine, by P. L. Mollison, M. D. (2nd edit.).